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The Massachusetts Medical Society

The next Annual Meeting of the Massachusetts Medical Society will be held in Boston, at the Hotel Statler, June 3, 4, and 5, 1935.

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NEW ENGLAND SURGICAL SOCIETY

SLIPPING EPIPHYSIS OF THE HEAD OF THE FEMUR*

BY JAMES WARREN SEVER, M.D.†

IN bringing this subject to your attention, I beg to advise you that I do so not because it is new or that I have anything of a startling nature to offer you. I do so largely in an attempt to rationalize the condition for you and for myself. The literature on this subject is increasing, but largely by the report of clinical cases; and a study of it leads, unless carefully analyzed, to confusion as to the best methods of treatment to be carried out.

ETIOLOGY: The etiology of this condition has caused great discussion, and is not settled yet. The three main causes are supposed to be: (1) Endocrine disturbance, (2) static forces, with which may be an undetermined low-grade infection, and (3) frank trauma. Any one of these conditions may predominate in any given case, or as far as one can tell, be the one determining cause of the epiphyseal slip. Secondary aseptic necrosis of the epiphysis, due to circulatory impairment from injury or infection, may well be a factor.

Endocrine disturbance or imbalance may be defined as abnormality of growth, overweight and height for the age, and a minus basal metabolism. On the other hand, several years ago, we thought of a slipped epiphysis as occurring only in the child with a definite endocrine imbalance, but with further observation and report of cases, we find many more occurring in the long and slender individual, or even in apparently normal children in whom no such condition exists. Trauma, or infection, in these latter cases, is undoubtedly the etiologic factor, although trauma per se is probably unusual as the sole cause. Trauma as a factor may easily be explained by the usual falls of children. One particular factor, in relation to trauma, may be of interest. Normally, extension of the hip is possible from 15° to 20°; beyond this point further extension is blocked by the tightening of the capsule and the strong anterior ligaments of the hip joint, and, also, by the fact that the head of the femur becomes securely locked in the acetabulum by extension. Therefore, upon forcible hyperextension at the hip joint, beyond these normal limits, any further motion must take place at the epiphyseal line, and the head of the femur may be slipped off the neck, or is partially separated at the epiphyseal line.

All this may be secondary to a primary weakening of the periosteum binding the head of the neck of the femur. The cause of this weakening is unknown, but is usually coincident with a period of unusually rapid growth during adolescence.[1]

Quoting Wilson[2], he states, "that experiments by Ollier, Poland and others have shown that the periosteum is the chief sustaining band between the epiphysis and diaphysis. Vogt and Kirmisson have suggested that the reason displacement occurs more readily in adolescents than in infants, is that, in the former, there are two bony surfaces glued together by a thin layer of cartilage, while in the latter, the epiphysis is a supple cartilaginous mass strongly bound by thick periosteum. In the case of the hip, the anatomic relation of the epiphysis to the femoral neck is potentially weak."

Another factor, which may have a bearing on the cause of the slipping, is that as the child grows older and approaches adolescence, the epiphyseal line, which in childhood is almost horizontal, tends to approach an oblique angle, in relation to the neck of the femur. Consequently, any increase in vascularity, and subsequent softening of the epiphyseal line, might result in a slip from the shearing forces of weight-bearing and undue strain.

As a matter of interest and importance more than half of the cases I have seen have been disabled at once by trauma of some sort, which of course may have been preceded by some epiphyseal lesion of which no history was obtainable. The trauma was real and not trivial. It is said, that there is no frank traumatic type of this disability, but one is led to doubt this statement, in view of such facts as these preceding immediate disability; being hit by an automobile, falling downstairs, tripping on car tracks, falling off a scooter, falling while roller skating, etc. It is also to be admitted that with preëxistent pain in the hip, and a limp, presumably from a congested epiphyseal line, a fall

*Read at the meeting of the New England Surgical Society, Burlington, Vt., September 29, 1934.

†Sever, James Warren—Associate Surgeon, Boston City Hospital and Boston Children's Hospital. For record and address of author see "This Week's Issue," page 1223.

may aggravate the condition and lead to an immediate and considerable slip. On the other hand, in this series, there are a number of cases with no known history of trauma.

PATHOLOGY: The pathology apart from the description of various specimens which have been removed for study is unknown. A study of all these various specimens shows various degrees of disturbance of the epiphyseal line and cartilage plate, with accompanying callus and necrotic areas, secondary to various degrees of displacement. But one case has been studied pathologically in the early or preslipping stage, when the original weakening of the epiphyseal line occurs.

C. Waltham Hospital; July 30, 1934; slight coxa vara, with some displacement of the epiphysis, following long-continued traction in bed, and later a plaster spica.

Ferguson and Howorth[3] reported in this instance that the capsule and periosteum about the neck were thickened, sclerosed and avascular, but that there was no evidence of rickets, infection or osteomalacia.

HISTORY AND ONSET: The history in these cases is usually one of rather insidious onset, in which trauma of varying degree, first or last, may or may not have been a factor in the picture. Many times the story is that the child has been aware of a dull gnawing pain in or about the hip, occasionally extending to the knee, and sometimes located just below Poupart's ligament. This may be, and usually is, accompanied by a slight limp. The child may have to go to bed from time to time to get relief from pain and discomfort. There may be definite periods of complete cessation from pain and symptoms, to recur after unusual activity.

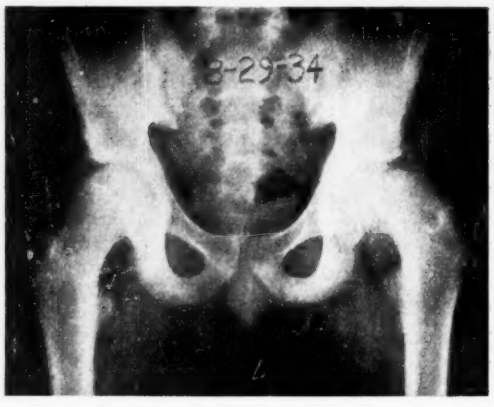
In a few cases, especially in the feminine types, the condition may affect both hips, either simultaneously, or, one after the other. This is a condition which should not be lost sight of, as it should be suspected and x-ray studies should determine its probability.

There are four stages of the condition recognized clinically:

(1) The preslipping stage.
(2) The stage where the epiphysis has slipped.
(3) The quiescent stage.
(4) The residual, or end result stage.

In the preslipping stage pain and limp are usual, associated with fatigue and disability with use. Examination then shows limitation of motion at the hip, especially in flexion and internal rotation. When the hip is flexed, it ability may occur. Increased pain, spasm, of the muscles may also be present. In the sectends to abduct and outwardly rotate. Spasm toms are more pronounced, and complete disability may occur. Increased pain, spasm, shortening and outward rotation of the leg occurs. In the first and second stages, increased hyperextension is an important sign to note. The quiescent stage, as the name implies, is that, where all symptoms have disappeared, and the residual stage, is that one, where, after treatment, the epiphysis has united with or without deformity.

X-RAY APPEARANCES: In the early, or preslipping stage, an x-ray shows a widened epiphyseal line, generally somewhat irregular, with already a tendency to widening or thickening of the neck, recently called coxa magnum. The



C. August 29, 1934; one year after onset, following many weeks in bed, with treatment by traction, showing slight tendency to coxa vara, slight slipping; functionally normal hip except for ½" shortening of the leg.

end of the neck, next to the epiphyseal line, may be irregular in outline, mottled and decalcified. The head does not show the mushrooming, or flattening, and fragmentation so often seen in Legg's disease, or coxa plana, and should be easily differentiated from that condition. The most usual diagnosis made, by those unfamiliar with the condition in the preslipping stage, is tuberculosis. An x-ray taken after the epiphysis has slipped, shows, of course, the lack of normal relation of it to the neck. The head is always displaced downward, inward and backward. In the later stages, with or without reduction, callus has formed along the line of separation, obliterating the epiphyseal line and

changing the apparent angle of the neck of the femur. This leads to the usual coxa vara deformity so often seen in varying degrees in the end results. Also, arthritic changes make themselves manifest comparatively early in the process of repair, with or without adequate reduction of the slipped epiphysis.

DIAGNOSIS: It is stated that pain in the region of the hip, associated with a limp, constant or intermittent, in an obese child, large for the indicated age, and with underdeveloped

M. October, 1933; right hip; aged nineteen, five years after onset. Coxa vara and slight displacement of the epiphysis. Epiphyseal line fused.

genital characteristics, should always make one suspect an injury to the upper femoral epiphysis. This is true, but many more cases, I believe, occur in children who are apparently physically within normal limits. Spasm of the hip muscles, pain in the hip or knee, limp, a sudden disability of the leg after a fall, all point to this condition. When the displacement of the capital epiphysis is more or less complete, the leg assumes the position of flexion, abduction and outward rotation, common to any capital fracture.

TREATMENT: In a series of twenty-two cases which I have collected this last year, and of which I have some complete and some incomplete records, only three were of the so-called obese and feminine types.

TREATMENT: The methods of treatment, I believe, have a very direct bearing on the end result, and I feel that in the last few years, distinct progress has been made along this line, which after all is the most important factor in the whole subject under consideration.

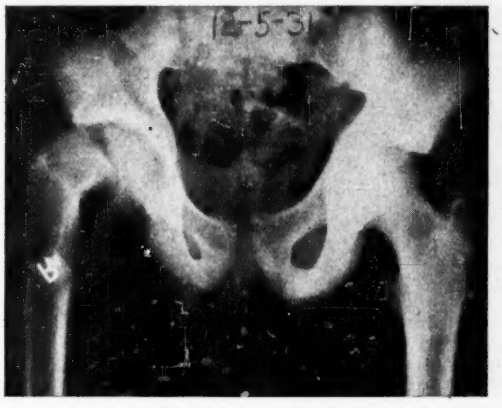
Considering treatment under the four headings stated above, namely: (1) The Preslipping Stage, (2) The Slipped Stage, (3) The Quiescent Period, (4) The Residual Stage, what should be the methods to be carried out in each stage?

THE PRESLIPPING STAGE: During this period not great damage has been done to the hip joint in terms of function. Occasional pain, limp and disability occur. This is the stage where the epiphyseal line is seen as irregular in the x-ray. It is congested, it is widened, and it takes but a slight external trauma or some misdirected mechanics to convert the condition into the second stage, that of the fully slipped epiphysis.

How can this be prevented?

First, by rest in bed; freedom from weight-bearing; traction on the leg to overcome pain, muscle spasm, and to prevent slipping of the epiphysis, which may occur even when turning in bed. One of my cases slipped her epiphysis as the result of the giving away of the traction, after it had been reduced by such a method and was supposedly held.

A spica, Thomas splint, with ambulatory



G. December 5, 1931; right hip. Note complete displacement of the epiphysis from the neck of the femur.

treatment is unwise, and unsafe in this stage.

Last year Jahss[4], in an attempt to save time, and the possibility of slipping occurring even with rest in bed, with traction or splints, in that stage, suggested that these hips be impacted, after the method of Dr. Cotton in fractures of the neck of the femur. He carried out this treatment with excellent results in three cases. The after-treatment consisted of a long spica for three months without weight-bearing. He felt, that, so traumatizing the epiphyseal line, it was possible to obtain premature ossification in the preslipping, and slightly slipped, cases. This seems a logical procedure, and should, I believe, be given further trial. In other clinics, drilling the epiphyseal line to establish premature ossification is practiced, but does not appeal to me as a conservative measure.

THE SLIPPED STAGE: Here we have to deal with two conditions, namely, that one, where the

slip is only about one-fourth to three-eighths of an inch, and that one where the slip is greater even to the complete separation of the head from the neck.

I believe, that, with only slight slips, seen in the early stages, that is in the first two weeks, rest in bed, and traction, are all that is necessary for a good result. Forcible manipulation in these cases generally does not work, and may, and often does, lead to considerable impairment of the motion and function.

Where the head has become fully separated, manipulation may restore the parts to their

N. October 11, 1934; end result eight years after onset, showing deformity of head; considerable arthritis about the hip joint; considerable limitation of motion; 1½" shortening.

normal relations. If this fails, and it generally does, an open operation, using the Smith-Petersen nail, as advocated and carried out by Wilson, may be tried. Another method, using open reduction, simply destroys the epiphyseal line surgically, followed by the re-implantation of the epiphysis. In this way union is assured, and foreign material is not left in the wound. In the cases of open operation, a certain percentage of stiff hips will result.

A word of caution about when to manipulate. If the condition or slip has existed six or eight weeks, in any degree, manipulation will probably fail to restore the fragments to their normal relationship, and a certain number will lose considerable motion and function as a result of such a procedure.

The use of a plaster spica after manipulation is essential. The leg should be fully abducted, internally rotated, and held in this position for at least two months, followed by a non-weight-bearing splint for several months.

THE QUIESCENT STAGE: During this stage, with or without a slip, there is not much spasm, and there is no pain. The condition is healing; the epiphyseal line is becoming obliterated but weight-bearing should not be allowed and the leg should be protected by a non-weight-bearing splint.

THE RESIDUAL STAGE: The residual stage generally presents a fused epiphyseal line, and a degree of coxa vara in direct relation to the amount of slip which was allowed to remain.

There is no treatment required except possibly a subtrochanteric osteotomy in an attempt to correct the coxa vara in part and so diminish the shortening of the leg.

As an end result condition, most cases pre-

L. July 17, 1934; x-ray shows preslipping stage in an endocrine type of a very obese boy. Left hip. Note mottling and absorption along the end of diaphysis.

sent one-half to three-fourths inches shortening, with slight limitation of motion especially in internal rotation and flexion at the hip. This may be considered a good result, either as the result of an open reduction, or as the result of traction and non-weight-bearing in the simpler cases.

The bare statistics of the twenty-two cases on which this paper is based, I will not present in whole to you, as they are of no particular interest. There are certain conditions, however, which are of interest, namely, thirteen boys to nine girls. Possibly trauma here may play a part. Average age thirteen years, the edge of adolescence, some as old as sixteen, one aged ten, who had had symptoms for a year. Variation in ages is consequently great, but running, roughly, between ten and sixteen. All known results had shortening from one-fourth to one and one-fourth inches.

The degree of deformity present in the final x-ray is not always to be considered an index of the degree of disability.

I have now discussed the points in relation to this condition which I wished to call to your attention, and which I believe should be stressed. I shall be greatly interested in your further discussion.

Name	Hip	Sex	Age at Onset	Type Endocrine	Duration First Seen	Treatment	Result	Supposed or Immediate Cause
Cahalane	Left	Male	13½	0	3 mos.	Bed with traction	¼" short Internal rotation	Various falls
Carlstrand	Right	Female	12	0	At once	Traction	½" short. coxa vara	Fall
Cataldo	Right	Female	12	0		Cast and then splint	¼" shortening Internal rotation and flexion limited	?
Cummings	Right	Male	13	0	1 mo.	Manipulation and cast	Not known	Fall
Emerson	Left	Female	13	0	Soon after onset	Splint	Coxa vara	Fell roller-skating
Gillio		Male	14	0	1 yr.	Manipulation and splint	Coxa vara	Fall?
Goulart	Left	Female	10	0	1 yr.	¾" shortening Manipulation and cast	Not known	?
Hansen	Left	Male	14	0	1 yr.	Manipulation and spica	1¼" short; poor	?
Krantz	Right	Female	12	+		Walking brace Manipulation and spica	1" shortening	?
Lavolo	Left	Female	12	0	At once	Cast	1" shortening; not good	Fall
Nestor	Left	Male	14	0	3 wks.	Cast and manipulation	Poor	?
Levine	Left	Male	12	+	2 wks.	Traction—splint	Good	?
Maguire	Right	Female	11	0	At once	Manipulation and cast	Not known	Fall
Marcus	Left	Male	13	0	10 mos.	Manipulation and cast	Good	Fall
Marotta	Right	Male	14	0	7 mos.	Cast six weeks Manipulation Bradford splint	Limited ¾" shortening Internal rotation	Skating accident
McLean		Female	13	+	6 wks.	Traction	No shortening; good	Fell down stairs
Schofield	Left	Male	16	Under-developed	At once	Cast and splint	Good	Fall
Sheehan	Left	Female	13	0	3 mos.	Spica	No shortening; good	Hit by auto
Thompson	Left	Male	16	0	1 mo.	Cast and splint	Good	None
Tubin	Right	Male	15	0	At once	Cast	Not known	?
Wright	Right	Male	12	0	At once	Manipulation	Limitation of motion in inward rotation and flexion; ½" shortening	
McFeeters	Right	Male	14	0	1 yr.	Manipulation, double spica	Stiff hip; permanent flexion 45°; 1¾" shortening	Two falls; immediate disability

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DISCUSSION

DR. FREDERIC J. COTTON, Boston, Mass.: *Mr. President and Members*—I hardly think it is in order for me to open the discussion because my experience has not been especially extensive. It has been one that has accumulated as the cases turned up in the Boston City Hospital.

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The only other point on which we don't quite agree is the matter of reduction of the slip cases. Years ago I saw a good many of these cases operated on by other people at open operation and I haven't particularly cared for the result. A nail is very likely to be left in the structure following the operation. This doesn't do any particular good.

I don't believe the reduction of these cases is quite so difficult as has been stated. If you take a case of this sort and reduce it carefully, using force, you must use a good deal of force. I haven't encountered any case I couldn't reduce approximately perfectly even if it had been slipped for a long time. It is rather interesting that the reduction of intracapsular hips, which I think everyone is beginning to accept, is on the same basis of manipulation we worked out many years ago on the slipped epiphysis; that is, a rotation and adduction of the hip with a bringing of the hip back into abduction with the maintained internal rotation, and that reduces the displacement of the necks in these cases, exactly as it does in the case of the fractured hips which we now know by reason of better x-rays.

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He has had an opportunity to have falls and injury to the epiphyseal line, and swelling there, but he gave no history of such trauma, and I think it would be called a one-accident case.

With respect to sequelae of these slipped epiphyses, we see many cases of arthritis in one hip, in adults, and some are secondary to slipped epiphyses in adolescence, but I wonder if others aren't the result of slight displacements that have healed without treatment and without recognition, with no more treatment than one would naturally give to a sore hip for perhaps a few weeks at a time. Having had a slipped epiphysis, this would set the stage by faulty mechanics, so that later in life perhaps the person would develop a very definite hypertrophic arthritis.

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In a study of 683 cases of the rheumatic infection in children whose histories were followed from five to twenty years after the onset of the disease, Findlay[3] found that 40 per cent died of heart disease within fifteen years and that as the older decades were approached an additional 30 per cent were incapacitated with an increasing number of deaths.

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is especially active in the joints, in the subcutaneous tissues and tendons, in the heart including the endocardium, myocardium, and pericardium, and in the brain. The infection becomes chronic with acute exacerbations. It is difficult to control and may retain its hold for years, manifesting itself periodically in one or the other of the acute forms.

Any one of these manifestations, rheumoidal arthritis, chorea, carditis including myocarditis, endocarditis, and pericarditis, and subcutaneous rheumatic nodules, may occur alone or in any order or combination. No one lesion should be considered the primary lesion. All are due to the same infection attacking susceptible tissues, but each results independently of the other. If one exception were to be made to this rule it would be that subcutaneous nodules do not occur alone or as the earliest evidence of rheumatism but are associated with extensive carditis and must be taken as an indication of serious myocardial disease. A case which recently came under my observation would seem to cast doubt on even this possible exception. The patient was a female child, eight years of age, who had no previous history of rheumatism. Numerous rheumatic nodules appeared on the child's head and extremities. One month later she had a typical attack of rheumatic fever with pain and swelling of the joints, elevated temperature, and precordial pain. One month following this attack she was admitted to the hospital. Upon examination she was found to have a slight enlargement of the pulmonary conus and a systolic murmur in the apex, which was transmitted to the axilla. During each of her three periods of hospitalization numerous rheumatic nodules were observed on her skull, spine, and extremities. Eighteen months after her first admission she died of myocardial incompetency.

There are three factors in the clinical picture of rheumatism which should be recognized. (1.) The disease may take a latent course for months, accompanied only by obscure and fleeting symptoms, such as "growing" or slight joint pains, a stiff neck, upper respiratory infections, tonsillitis, pharyngitis, or a brief indeterminate febrile condition. When an acute attack of rheumatism occurs accompanied by pain, the child is usually brought to the attention of a physician. But when, as frequently occurs, the infection is active but not overt, extensive damage may result before the symptoms are sufficiently acute to command attention. Some of the most severe types of rheumatic heart disease which I have observed were those in which the symptoms had been either absent or so slight that a physician was not consulted until the patient was suffering from cardiac incompetency.

(2.) Carditis may the first acute manifestation of the disease. It is in these cases that cardiac incompetency may be well established before medical advice is sought. Although it is unusual for an initial attack of acute rheumatic carditis to terminate in death, it sometimes occurs when early treatment is not instituted. One such case that came under my observation was that of a male child, four years of age. He had been breathing with difficulty for a month previous to his admission to the hospital, and for two weeks had coughed a great deal, vomited and complained of pain in the epigastrium. No history of rheumatic infection could be elicited. Upon examination a diagnosis was made of acute rheumatic heart disease with dilatation and myocardial incompetency. The patient died three months after admission to the hospital. The autopsy revealed extensive inflammatory infiltration of the myocardium characteristic of rheumatic heart disease.

(3.) The incidence of heart disease in rheumatic children indicates that the heart is especially susceptible during childhood to the rheumatic infection. Findlay[3], in a study of 108 rheumatic children examined prior to the appearance of carditis, found that 42 per cent developed heart disease during the initial attack of rheumatism and that in 43 per cent of the remaining number the heart was involved during the second attack. Three of the 108 children passed through five attacks each without developing carditis. These findings show that the individual susceptibility to the rheumatic virus varies, that the heart is damaged in a majority of the cases of rheumatism in children, and that the damage usually occurs early in the course of the disease. It is a common observation that once the heart is involved each successive exacerbation of the disease may extend the damage.

A considerable variation is found in the extent to which the heart may be involved during an acute attack of rheumatic carditis and in the amount of permanent damage remaining after its subsidence. In determining the factors responsible for the progress of an individual case it is impossible to gauge the relative importance of the virulence of the infecting agent, the individual's susceptibility, and the promptness with which treatment is instituted.

In the group of cases, however, in which the disease is promptly recognized and treatment is begun early, an incipient rheumatic infection may often be controlled before the myocardium or the valve leaflets become extensively affected. A mild attack of myocarditis may subside without leaving permanent damage or functional disturbance. I have often observed from month to month the gradual return of the heart to its normal size and the frequent early disappearance of murmurs.

In a second group of patients the heart mus-

cle is more seriously involved and some degree of permanent change results. As these children are examined from time to time a persistent systolic murmur is found in the mitral area. Even with this murmur well established, symptoms of cardiac incompetency may be entirely absent provided there is no accompanying enlargement of the heart. These patients, unfortunately, seldom reach maturity without recurrent rheumatic infections.

There is a third group of children who continue to show symptoms of cardiac distress indicative of extensive and permanent changes in the heart muscle. In many cases of this group the damage has been cumulative. One frequently views the tendency of the rheumatic heart to become more seriously affected with each recurrence of rheumatic fever, rheumatic arthritis, or chorea. In other cases the damage, even when extensive, is the result of a single attack which was unusually prolonged either because of the intractable virulence of the infection or because it took a latent and undetected course and treatment was delayed.

A fourth group of cases has been noted which may be called the chronic relapsing type. In these patients frequent relapses result in progressive carditis and the development of permanent cardiac incompetency. This chronic or relapsing type of carditis is one of the most intractable forms with which we have to deal. It leads to permanent and extensive changes in the myocardium, which are first inflammatory, then exudative and later fibrotic. Pericarditis, at first inflammatory, then exudative and later adhesive, is frequently seen in patients included in this group.

When, during the course of an active carditis, there is a sudden exacerbation of the disease accompanied by increased respiration, elevation of temperature, fretfulness, and languor, pericarditis may be suspected. The patient may have been progressing satisfactorily with symptoms of carditis disappearing, the size of the heart receding, the pulmonary congestion clearing, and the dyspnea lessening. With the development of acute pericarditis, however, there is a lighting up of the whole process. The temperature is suddenly elevated, the heart rate accelerated, dyspnea reappears, and there may be prostration, precordial and abdominal pain, and vomiting. An attack of acute pericarditis may simulate acidosis, toxemia or an acute surgical condition of the abdomen.

The advent of pericarditis may change the picture from day to day as the various areas of the pericardium become inflamed and subside, only to have new areas become involved in the process. Pericardial fluid in large enough amounts to be demonstrated does not necessarily appear, nor does the continuing process necessarily result in adhesive pericarditis in mass. The inflammation may subside and leave but occasional pericardial adhesions, or in some instances none whatever. When adhesive pericarditis has become chronic and is associated with mediastinitis and union of the outer layer of the pleura and the chest wall, we are confronted with one of the most serious forms of cardiac disease, particularly in early life. In these instances the peritoneum may be involved, with an accompanying perihepatitis, cirrhosis, and ascites (Pick's syndrome).

The development of acute pericarditis in rheumatic heart disease makes the ultimate outlook less favorable. It is generally conceded that the heart's efficiency depends upon the degree of myocardial reserve, and because of the close proximity of the pericardium and myocardium, the inflammatory process of acute pericarditis will of necessity extend the myocardial damage.

Acute rheumatic carditis may be demonstrated clinically by an acceleration of the heart rate, dilatation, and distinct intracardial murmurs. A systolic murmur at the apex may be the first symptom of carditis in acute rheumatic fever. As the carditis becomes more pronounced and the heart enlarges, the murmurs increase in their intensity. Experience has shown that murmurs attributed clinically to pathological valvular defects may, in some cases, be due to myocarditis or to dilatation of the heart. I have observed in many instances that the murmurs accompanying dilatation of the heart disappear with the return of the heart to its normal size. I have also observed instances in which physical signs descriptive of both mitral stenosis and aortic regurgitation accompanied dilatation of the heart, and at autopsy no gross pathological changes of the valves were present. One such case may be cited of a male child, eleven years of age, who was under my observation for two and a half years with a diagnosis of rheumatic heart disease, mitral stenosis and incompetency, aortic stenosis and incompetency, and cardiac dilatation. At autopsy no gross changes were present in the valve leaflets. Even with pathological valvular defects present, the prognosis and the curtailment of the patient's activity must depend mainly upon the physical signs and symptoms which give evidence of the loss of reserve power of the heart muscle, rather than upon the murmurs per se. The murmur most frequently heard in children is the systolic murmur at the apex. But unless there has been a recent attack of rheumatism or chorea, or there is present an enlargement of the heart or a lack of response to effort, the systolic murmur at the apex has no significance.

Electrocardiographic study of the rheumatic child may or may not demonstrate important findings during an acute attack of carditis. If electrocardiographic changes are present during

a first or second attack, it does not necessarily mean that the prognosis is unfavorable. In a first or second attack, the arrhythmias, such as premature auricular and ventricular beats, dropped beats (sino-auricular block), and transitory or changing auriculoventricular block, may in some cases appear momentarily, due to a toxemia of the conduction system, which has developed from the bacterial or disturbed metabolic processes. When unchanging heart block is demonstrated in the initial electrocardiographic studies of a young child, it is possible that it may be of congenital origin.

The clinical, pathological, and electrocardiographic studies in the following four cases present various significant phases of rheumatic heart disease.

CASE 1. A male, aged four years, was admitted to hospital May 14, 1929, with a history of having been "ill off and on" for two months and having had "a touch of pneumonia" two weeks previously. Otherwise his history was irrelevant.

On admission the temperature was 100°F., the heart rate 98, and the respirations 28 per minute. The heart was enlarged in all diameters. There was a diastolic and a systolic murmur at the apex. A systolic murmur in the aortic area was also present. A few medium râles were noted in the bases of both lungs. There was slight enlargement of the liver. The urine was essentially negative. The blood Wassermann test was negative. The hemoglobin was 56 per cent, the red cells 3,720,000, and the white cells 17,800, of which 22 per cent were lymphocytes and 78 per cent polymorphonuclears. A working diagnosis was made of acute rheumatic heart disease, with acute dilatation and acute pericarditis.

The patient rapidly developed advancing signs and symptoms of congestive failure and of massive pericardial effusion, and died two months after admission. Six days before death occurred, an electrocardiogram was taken which demonstrated a complete auriculoventricular dissociation (fig. 1). An autopsy was not permitted.

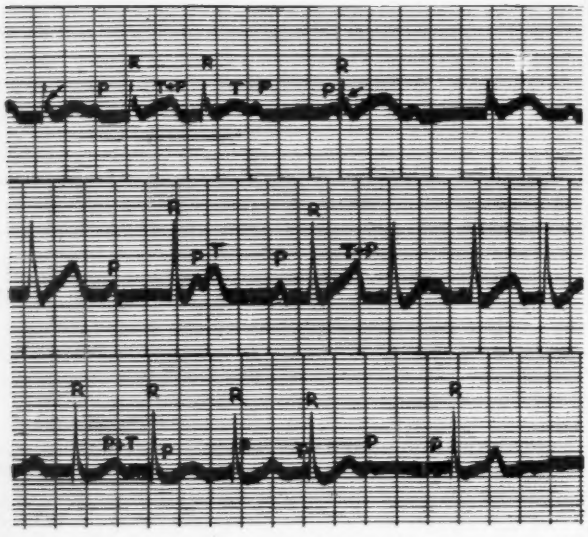

FIG. 1. Case 1. Male, aged 4½ years. Acute rheumatic heart disease. July 11, 1929. Rate: 100 A-V. Axis: +70°, normal. Complete auriculo-ventricular dissociation. Notching of the R wave in lead I.

CASE 2. A female, aged seven years, was admitted to hospital on July 16, 1929, with a history of pyrexia, coughing, and acute pain in the joints and epigastrium of two weeks' duration. She had had a similar attack two months previously. Physical examination showed that the heart was enlarged in all diameters and that a systolic and a diastolic murmur were present at the apex. A few medium râles were noted in the bases of both lungs. The liver was not enlarged. A diagnosis was made of acute rheumatic fever with carditis. The child improved rapidly and was discharged two weeks after admission.

During the following four and a half years the patient had recurrent attacks of rheumatic arthritis with pyrexia. Acute catarrhal bronchitis was a frequent accompaniment. Two years after her first admission she was again admitted with well-developed signs of cardiac incompetency. An examination of the urine showed a few squamous epithelial and white blood cells. The blood Wassermann test was negative. The hemoglobin was 41 per cent, the red cells 3,750,000, and the white cells 14,400, of which 21 per cent were lymphocytes and 79 per cent polymorphonuclears. She remained in the hospital for five months and then was discharged, only slightly improved, because of nostalgia.

On April 20, 1933, one year following her second discharge, she was readmitted. An electrocardiogram taken the following day showed a split and rounded P wave in leads I and II, suggestive of involvement of the auricles. There was also a notching of the descending limb of the R wave in lead I (fig. 2). At this time the patient was showing clinical signs and symptoms of advanced congestive failure. After a two-months' residence in hospital she was discharged, and six months later was readmitted. The following day she died. An electrocardiogram demonstrating auricular fibrillation and inversion of the T wave in lead I was taken two hours before death (fig. 3). At autopsy numerous petechial spots were observed in the epicardium. The myocardium was flabby and lighter than normal. The endocardium showed no evidence of acute inflammatory reaction. The tricuspid leaflets, however, appeared somewhat thickened, and the endocardium above the mitral valve showed some athero-

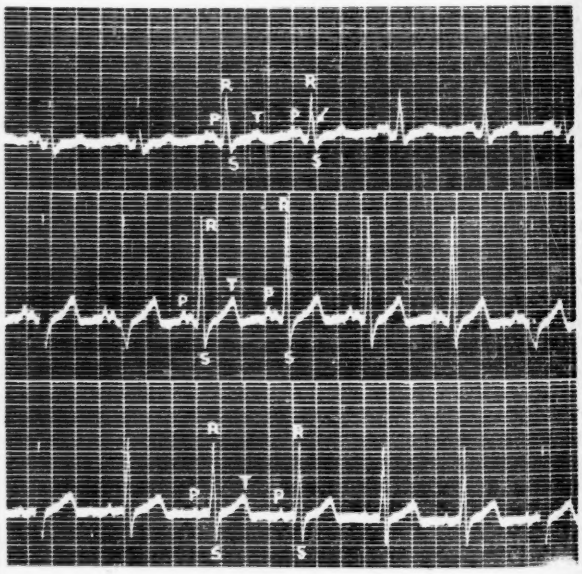

FIG. 2. Case 2. Female, aged 7 years. Acute rheumatic heart disease. April 21, 1933. Rate: 100 A-V. Axis: +66°, normal. P wave in leads I and II split and rounded, indicating dilatation of the auricles. Notching of the R wave in lead I.

matous degenerative changes. An anatomical diagnosis was made of chronic adhesive pleuritis, bilateral; pleural effusions, bilateral; pulmonary congestion and edema; bronchopneumonia, lower lobes, bilateral; chronic adhesive pericarditis; chronic myocarditis; dilatation of the heart with insufficiency

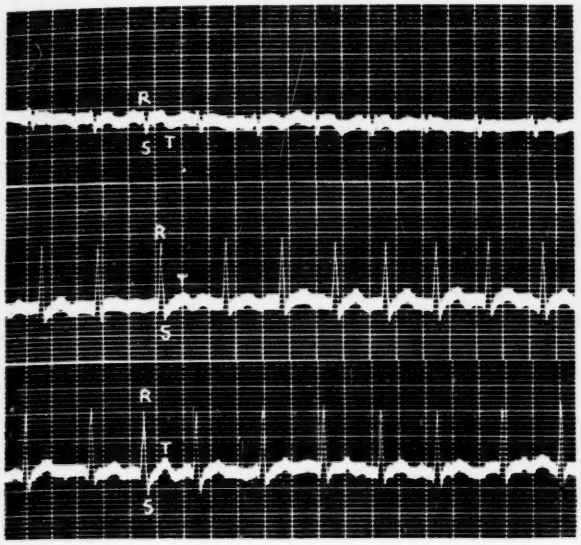

FIG. 3. Case 2. Taken of the same patient as in fig. 2, 7½ months later, December 2, 1933, and 2 hours before death. Rate: 150 Ventricular. Axis: +96°, R. A. D. Auricular fibrillation. Inversion of the T wave in lead I.

of the mitral, pulmonic, and tricuspid valves; chronic valvulitis, tricuspid, healed; abdominal ascites; peritoneal adhesions; chronic passive congestion of the liver, spleen, and kidneys. It is interesting to note in this case that stenosis of the mitral valve was not present, although on physical examination a definite diastolic murmur was heard.

CASE 3. A male, aged nine years, was admitted to hospital on June 12, 1930, with a history of attacks of vomiting over a period of six weeks just prior to admission. He was dyspneic and orthopneic. His heart was enlarged in all diameters. A systolic murmur was present at the apex. Numerous moist râles were elicited throughout both lungs. The liver was enlarged. Ascites was present. The urine was essentially negative. The blood Wassermann test was negative. The white cells were 12,500, of which 23 per cent were lymphocytes and 77 per cent polymorphonuclears. A diagnosis was made of acute rheumatic heart disease, with cardiac incompetency and acute dilatation.

The patient improved and was discharged from hospital three months after his admission. On February 17, 1931, five months later, he was again admitted because of coughing, dyspnea, and ascites. The heart was enlarged in all diameters. The systolic mitral murmur in the apex, previously noted, remained present. There were dullness and moist râles in both lungs. The liver was enlarged. An electrocardiogram taken February 21, 1931, demonstrated an auriculoventricular rate of 80 beats per minute with premature ventricular contractions, bigeminal (fig. 4). The child improved rapidly and was discharged March 14, 1931, one month after admission. It is interesting to note that at no time during his four months of hospitalization were physical signs of rheumatic fever or rheumoidal arthritis present. For the past three years he has been examined at intervals in our cardiac clinic, and although the systolic murmur in the apex persists there have been no physical signs of active rheumatic carditis.

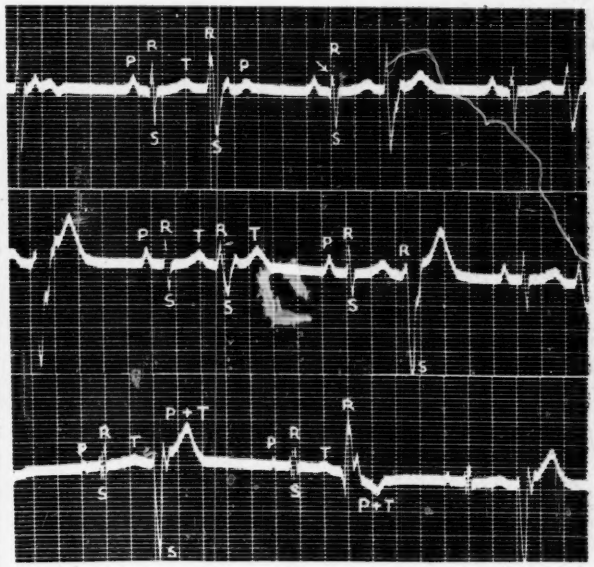

FIG. 4. Case 3. Male, aged 9 years. Acute rheumatic heart disease. February 21, 1931. Rate: 80 A-V. Axis: —150°, L. A. D. Premature ventricular beats, bigeminal. Notching of the R wave in lead I. R-S-T complex bizarre in lead III.

CASE 4. A female, aged four and a half years, was admitted to the hospital with a history of sore throat, coughing, restlessness, and pyrexia of one month's duration. On admission the child was dyspneic and orthopneic. The temperature was 101°F., the heart rate 100, and the respirations 25 per minute. The heart was enlarged in all diameters. A systolic murmur was present at the apex. A few medium râles were heard in the bases of both lungs. The liver was enlarged. The urine was essentially negative. The blood Wassermann test was negative. The hemoglobin was 56 per cent, red cells 3,260,000, and white cells 15,200, of which 35 per cent were lymphocytes and 65 per cent polymorphonuclears. A diagnosis was made of acute rheumatic heart disease, with acute dilatation and acute pericarditis.

The patient improved satisfactorily and was discharged two months after admission. One year later, on February 24, 1930, she was readmitted because of attacks of coughing and vomiting of two weeks' duration. The temperature on admission was 102.6°F. and the heart rate 96 beats per minute. The heart was enlarged in all diameters, with an enlargement of the pulmonary conus. A systolic and a diastolic murmur were present at the apex. A pericardial friction rub was noted. Fine and medium râles were present in both lungs. The liver was enlarged. Two months after admission she was discharged improved.

On October 7, 1930, six months after discharge, she was again admitted with signs of advancing cardiac incompetency. She was digitalized, beginning October 23, 1930, and was then continued on a maintenance dose of grains 1½ of the powdered leaf. On December 22, 1930, an electrocardiogram demonstrated a digitalis T wave (fig. 5).

The patient was discharged on January 13, 1931. On February 14, 1931, she was readmitted and died one month later. At autopsy an examination of the pericardial sac showed a parietal layer firmly adherent to the visceral layer. The walls of both ventricles were thin. There were small areas of fibrous

thickening in the myocardium. The leaflets of all the valves were in good condition except the anterior leaflet of the mitral valve, which was narrowed. An anatomical diagnosis was made of chronic

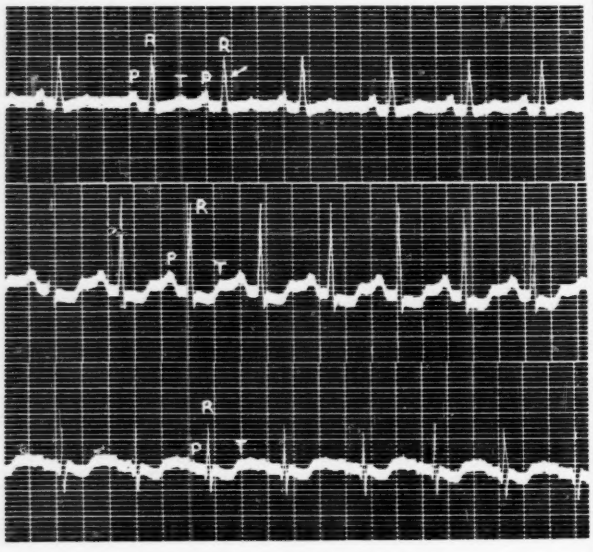

FIG. 5. Case 4. Female, aged 5 years. Acute rheumatic heart disease. December 22, 1930. Rate: 110 A-V. Axis: +54°, normal. Digitalis T wave in leads II and III. Notching of the R wave in lead I.

adhesive pleuritis, bronchopneumonia and pulmonary congestion, chronic adhesive pericarditis, myocarditis, and chronic passive congestion of the liver, spleen, and kidneys.

TREATMENT.—The treatment of rheumatic heart disease in children rightly begins with the early and adequate treatment of the rheumatic infection. The serious cardiac disability which often follows what has appeared to be a mild and fleeting attack of rheumatism makes it imperative that every manifestation of the rheumatic infection, however slight, be treated immediately. The administration of the salicylates should unquestionably be initiated whenever any of the forms of the rheumatic infection manifest themselves. Although there is no evidence that the administration of the salicylates will modify the course of chorea, subcutaneous nodules, or carditis, there is unmistakable evidence that in the treatment of rheumatic fever and rheumoidal arthritis the salicylates when given in sufficient quantities are of definite therapeutic benefit. There is also evidence that when acute rheumatic fever and rheumatic arthritis are adequately treated with the salicylates, carditis occurs less frequently than when they are not so treated. Therefore, until the rheumatic virus can be isolated and studied and a specific remedy be evolved, the salicylates should be used in the treatment of all of the manifestations of the rheumatic infection. After the subsidence of symptoms it is advisable as a prophylactic measure to continue for a time the monthly administration of the salicylates for periods of ten days. It is also advisable in all cases of established rheumatic heart disease to give rations of the salicylates three times a day over a ten-day period monthly during the autumn, winter, and spring, and always during the stages of acute upper respiratory infections.

A minimum dose of fifteen grains each of sodium salicylate and sodium bicarbonate should be given three times a day after meals. Much of the doubt growing out of the administration of the drug is due to the insufficient dosage employed. It is not unusual to have a relapse of rheumatic fever or of rheumoidal arthritis develop while the patient is receiving from 20 to 30 grains of sodium salicylate after each meal, and its subsidence brought about by increasing the dose to approximately 40 grains three times a day after meals.

It must be remembered that the administration of sodium salicylate in large doses may produce acidosis, and that an alkali must be given simultaneously in amounts equal to or greater than the salicylates. The value of combining an alkali with the salicylate has frequently been questioned. Morris and Graham[4] offer the explanation, based upon physiologic tests which they conducted, that sodium salicylate by impairing the function of the kidneys allows the acid bodies to accumulate; whereas when an alkali is combined with the salicylate the kidneys are able to maintain their function as indicated by an increased output of nitrogen, chlorine, and water, as well as by a greater excretion of sodium salicylate. It has been my practice recently to use an alkalined salicylate (salicionol). If sodium salicylate is administered in combination with an equal or double quantity of sodium bicarbonate, and in addition if the patient has a daily evacuation, the danger of acidosis occurring is minimized. When, however, sodium salicylate is being administered in large doses over a long period of time, the symptoms should be closely watched and the urine examined frequently for diacetic acid and acetone. If symptoms of acidosis appear, the discontinuance of the drug for twenty-four hours and the administration of a purgative will usually relieve the condition. Sodium salicylate administered in large doses may also cause the red corpuscles to break down rapidly.

In the treatment of all the manifestations of the rheumatic infection, absolute rest should be instituted and sufficiently prolonged. When there are definite signs of active carditis, a rest period in bed of six months or longer may, in a first or second attack, restore the normal function of the heart. If the first manifestation of the rheumatic infection is properly and thoroughly treated, a large percentage of the patients will recover from their rheumatism without cardiac involvement; and if the heart does become involved, all physical signs of the involvement will, in many cases, subside. The thorough treatment of a second attack of rheu-

matic carditis may also result favorably, but subsequent attacks as a rule cannot at present be treated with any great degree of success.

It is important in treatment of active rheumatic carditis to prolong the period of rest in bed and of the salicylate therapy to a time safely past the complete subsidence of the active phase of the infection. It is at present impossible either by physical signs or laboratory tests to determine precisely when the inflammation has entirely subsided. The erythrocyte sedimentation rate may be of importance in determining when the infection in cases of rheumatic carditis has become inactive, but sufficient work has not been done to justify confidence in the method.

The diet should be nourishing, high in carbohydrates, and easily digested. In the active stage of the disease frequent feedings should be given in small amounts. When the patient is showing passive congestion of the lungs and liver, ascites, edema of the face and extremities, and a decreased urinary output, the Karrell diet is of special value and should be given over a period of five to six days and on two successive days of each week until the passive congestion has subsided. When there is retention or edema, the fluid intake should be restricted. In the congestive state of rheumatic heart disease, the quantity of fluid intake and of urine excreted should be observed and recorded. It is not advisable to allow the urinary output to fall below 50 per cent of the fluid intake. The intake may be controlled, and when the quantity of urine excreted is being reduced too far below the intake, I have observed that the administration of ammonium chloride in sufficient doses will produce diuresis. It is my practice to administer it in gelatine capsules covered with a medically inert substance which is not acted upon by the gastric juices but is acted upon by the alkaline media of the intestinal tract.

Secondary anemia is an almost constant accompaniment of active rheumatic carditis and should receive appropriate treatment. When and if the hemoglobin falls to 50 per cent or below, periodic blood transfusions of 100 to 200 cubic centimeters have, in my experience, been followed in many instances by marked improvement.

The removal of carious teeth and infected tonsils is by no means a cure for the rheumatic infection, nor will it prevent or minimize the severity of future attacks. The procedure is justified, however, because of the improvement in the general health of the children observed following the removal of foci of infection. If a tonsillectomy or a dental extraction is decided upon, the procedure should be instituted only after the subsidence of the active stage of the disease. An alkalined salicylate or sodium salicylate and sodium bicarbonate in moderate doses should then be administered three times daily for ten days prior to extraction or tonsillectomy and continued for a period of ten days following the procedure, as a prophylactic measure. When both a tonsillectomy and an adenoidectomy are advisable, the adenoidectomy should be deferred until after the healing following the tonsillectomy is complete.

Pharyngitis in the rheumatic child has, in many instances in my experience, been the forerunner of a renewed attack of carditis. I have repeatedly noted this association. Every precaution should, therefore, be taken to protect the patient from extreme changes of temperature, from dampness, cold, and draughts. He should be absolutely isolated from individuals suffering from upper respiratory infections. Bronchopneumonia is, our records show, unfortunately a frequent and sometimes fatal accompaniment of rheumatic carditis. Children having rheumatic carditis should constantly wear a light woolen undergarment during the winter months as an additional protection, and during the period when they are in bed with acute attacks they should at all times wear a woolen garment covering the chest.

Digitalis therapy, it has been observed, has not always been administered in the present accepted form or dosage. Very good results are obtained in selected cases by the use of a standardized powdered leaf of digitalis administered according to the method of Eggleston[5]; that is, 1½ grains given for every ten pounds of body weight, plus 1½ grains per day during the period of digitalization to compensate for the amount eliminated in the twenty-four hour period, after which 1½ grains given daily as a maintenance dose. As occasion arises three grains may safely be administered on alternate days, provided that the dosage is controlled by frequent electrocardiographic readings. The administration of digitalis in sufficient amounts to reduce the heart rate in cases of carditis without frequent observation of the electrocardiographic tracings may prove disappointing. I have observed many cases of heart block in children during the course of their active rheumatic carditis. It is now generally conceded that children will tolerate larger doses of digitalis per body weight than will adults. The patient's optimum dose must be determined. In the study of a large number of electrocardiograms of children who were receiving powdered digitalis leaves as a therapeutic measure and who, subsequent to digitalization, were being continued on a maintenance dose of 1½ grains of the powdered leaf. I have frequently noted that no change was brought about in the P-R interval or in the R-T or S-T segments. In quite a few instances 3 grains given on alternate days were necessary to develop a digitalis effect in the electrocardiogram and to bring about clinical improvement. I

have likewise observed that there is less gastric disturbance with the administration of the powdered leaf in medically inert coated capsules. I have had no experience in administering digitalis to a child under four years of age.

In the acute stage of rheumatic carditis with an elevation of temperature, rapid heart action, and the presence of arrhythmias such as changing blocks and extrasystoles, digitalis must be withheld, even though the accompanying signs and symptoms of cardiac insufficiency and acute passive congestion are present. In these instances theocalin and the salicylates are the drugs of selection. It has been my experience that all cases of active rheumatic carditis respond more favorably when treatment is initiated early by sufficient doses of the salicylates and by absolute rest in bed.

This dictum does not apply, however, to the cardiac patient who has passed the active stage of his rheumatic disease. When and if the active manifestations of the rheumatic infection subside and there is still dilatation, passive congestion of the lungs and liver, and an absence of changing heart block, digitalis has a rightful place in the treatment. Its use is especially indicated in cases in which auricular fibrillation has developed. It is in these two conditions that digitalis has its greatest value.

SUMMARY

1. Heart disease in patients under twenty years of age is, with few exceptions, rheumatic in origin.

2. The heart in childhood is especially susceptible to rheumatic infection. Carditis may be the first manifestation of the infection.

3. Rheumatic heart disease in its early stages may not be accompanied by overt symptoms and may go unrecognized until signs of cardiac incompetency appear.

4. The systolic murmur at the apex, frequently found in children, is without significance unless there has been a recent attack of rheumatism or chorea, or there is present cardiac dilatation, rapid heart action, or a lack of response to effort. The curtailment of the child's activity depends upon the lack of response to effort and not upon the presence of the murmurs.

5. Effective treatment of rheumatic heart disease begins with the early and thorough treatment of the first manifestation of rheumatic infection and includes adequate precautions against subsequent attacks.

6. When the treatment of rheumatic carditis is instituted early and is sufficiently prolonged, the attack may subside without leaving permanent damage or functional disturbance.

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COARCTATION OF THE AORTA IN FATHER AND SON

BY WILLIAM G. WALKER, M.D.†

HAVING found no mention of coarctation of the aorta in lineal descendants in the comprehensive papers at hand[1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12], the discovery of the anomaly in a father and his son prompted this report.

The first patient was seen several times during a period of ten years but the cause of his disability was not recognized until the end of this time. The second patient, the son of the first, was seen twice in 1931 but the signs of coarctation were overlooked, and it was not until he was seen in July, 1934 that the correct diagnosis was made.

CASE REPORTS

CASE 1. Albert D., a business executive, aged forty-eight years.

He was first seen on December 11, 1924, at the age of thirty-nine. He complained of feeling "all in". This feeling came on five years before, after hard manual labor and considerable business worry, and has remained a constant symptom since then. Hypertension had been found at an insurance examination two months previously. For one year he had experienced dyspnea upon exertion, and palpitation and pain around the heart following exercise.

The only interesting feature in the family history was that a sister, who had never been well, had "heart trouble". She died at thirty-five of pneumonia.

Physical examination revealed a well-nourished, white man, with gray hair. There was slight variation in the calibre of the retinal arterioles. The teeth showed evidences of much dental work. The tonsils had been removed. The pulse rate was twenty to the quarter minute. The right radial wall was moderately thickened and slightly tortuous. The blood pressure* was 186 systolic and 108 diastolic. The apex impulse and the left border of dullness of the heart were 10 centimeters to the left of the midsternal line. The right border of the heart was 2 cm. to the right of the midsternal line, and the supracardiac dullness measured 3 cm. The cardiac rhythm was regular. A systolic murmur was heard over each valve area, being loudest at the second right interspace. This murmur was transmitted to the right side of the neck. The lungs were clear, and no abnormalities were found in the abdomen. There was slight pretibial edema.

Laboratory Examination: The hemoglobin was

†Walker, William G.—Physician to The Brockton Hospital since January, 1924. For record and address of author see "This Week's Issue," page 1223.

*All brachial blood pressures, unless otherwise stated, were taken on the right arm. An ordinary blood pressure cuff was used for all determinations.

100 per cent by the Tallqvist method. The urine was essentially negative and remained so at several examinations, save for the appearance of a moderate number of hyaline casts in June, 1930, following the use of potassium sulphocyanate. The blood Wassermann was negative.

The diagnosis of essential hypertension was made, and he was advised to obtain more rest and recreation.

During the next year and a half he was seen at various times. He complained mostly of lack of energy. He also was troubled with attacks of mental confusion and "ringing" in the head, gaseous eructations, and constipation. Palpitation was troublesome, and premature beats were noted at most of the examinations. The slight pretibial edema persisted for some time but later disappeared.

The blood pressure readings obtained throughout the period of observation are given in table 1.

TABLE 1

B. P.	12/11/24	3/27/25	11/12/25	12/10/25	10/2/26	3/25/30	4/2/30	5/1/30	5/24/30
	186	*154	152	178	182	206	202	†192	180
	108	90	76	88	106	106	106	96	102
B. P.	6/26/30	7/18/30	7/26/30	9/8/30	6/27/31	7/2/34			
	‡158	172	150	158	164	158			
	98	88	90	92	90	82			

Each blood pressure reading represents the lowest systolic and diastolic pressure found in three or four consecutive estimations.

*This reading followed an extended vacation in the South.
†This reading followed four weeks' rest in bed.
‡Has been taking 4½ grains of KCNs daily.

When examined in November, 1925 it was noted that the persistent systolic murmur was loudest at the fourth left interspace close to the sternum.

In March, 1930 his basal metabolic rate was minus 8 per cent. During the same month gastric analysis showed on the fasting contents: free hydrochloric acid 30. Combined acid 73. One hour after a test meal free hydrochloric acid was 34 and combined acid 75. Fluoroscopy of the chest and gastro-intestinal x-ray and gallbladder studies at this time were reported as negative. The phenolsulphonphthalein excretion was 70 per cent in two hours and ten minutes.

In March, 1931 he passed a large amount of mucus and some blood by rectum. Digital and proctoscopic examination revealed only a lone hemorrhoid. Later the same year he had severe attacks of aching in the abdomen. Nothing was found to account for them but the patient ascribed them to the proctoscopic examination.

Following an absence of three years the patient returned on July 2, 1934. Because of business reverses he was doing manual labor. He was still short of breath, occasionally had "ringing" in his head, and felt tired all the time. For the past few years he had noticed weakness and stiffness in the knees after sitting. He was quite spare, having lost about twenty pounds since 1924. The apex impulse of the heart, and the left border of cardiac dullness were 9.3 cm. to the left of the mid-sternal line. The left clavicle measured 17.2 cm. The cardiac rhythm was irregular, due to a moderate number of premature beats. With the patient recumbent a systolic murmur was heard in the third and fourth interspaces on either side close to the sternum. This was loudest at the third left interspace. It was also heard over the second right interspace close to the sternum. The murmur was transmitted to the right side of the neck. In the erect position the murmur was heard over the third and fourth interspaces on the right but it was not heard over the aortic area or to the left of the sternum. A systolic murmur was heard on either side of the spinous processes of the upper dorsal vertebrae, above the interscapular region, being better heard on the right side. Large, tortuous, visibly pulsating subscapular arteries were seen in the posterior axillae, while below the inferior angle of the right scapula a pulsating vessel was seen, and in the same region on the left side 8 cm. below the left scapula was a raised area, approximately 2.5 cm. in diameter and 4 mm. in height, in which tortuous, pulsating vessels were felt. No pulsation could be felt over the abdominal aorta. The femoral, dorsalis pedis, and posterior tibial pulses were weak. The right brachial blood pressure was systolic 158, diastolic 82, and the left femoral blood pressure was 108 systolic, 94 diastolic.

At fluoroscopic examination of the chest by Dr. L. B. Packard, the ascending and transverse aorta pulsated vigorously. Just below the aortic knob no pulsation was seen, and the aortic shadow appeared indented.

Figure 1 is a reproduction of a posterior-anterior x-ray film of the chest which reveals the typical scalloping of the ribs posteriorly. The prominent aortic knob shown here is unusual in this condition. The scalloped areas have been retouched.

CASE 2. Ellery D., aged 18, son of Albert D.

When first seen in March, 1931, at the age of 15, he complained of being "all run down". He said that he had always been nervous and high-strung. Colds, frontal headaches, and middle-ear infections had been frequent. Dizziness was noticed at times upon suddenly arising in the morning. Dyspnea was induced by little sustained effort, and palpitation developed if he ran. His appetite was good, and he had gained six inches height in the past two years. The tonsils and adenoids had been removed when he was six years old. On several occasions incision of one or the other ear drum had been done for otitis media. One year ago he had been confined to bed for several weeks because of an acute mastoiditis.

Physical Examination: The patient was pale and thin. He measured 5 feet, 11¾ inches (182.5 cm.) in height and weighed 132½ pounds. (60 Kg.) The pupils reacted to light. The optic discs were sharply outlined, and the retinal arterioles were narrow and tortuous. There was almost complete obstruction of the right naris. The teeth were irregularly

placed. The blood pressure was 142 systolic, 80 diastolic. The apex impulse and left border of cardiac dullness were just outside the left nipple line. The rhythm was regular. A systolic murmur was heard at the apex. The lungs and abdomen revealed no abnormalities. The reflexes were normal.

Laboratory Examination: The hemoglobin was 105 per cent by the Sahli method. The urine showed a cloud of albumin with heat and acetic acid. Otherwise it was negative. The phenolsulphonphthalein excretion was 47 per cent in two hours and ten minutes. When the test was repeated three months later there was an 80 per cent output of the dye. The blood Wassermann and Kahn tests were negative.

In June, 1931 the blood pressure was systolic 156, diastolic 102. The relative cardiac dullness was 13

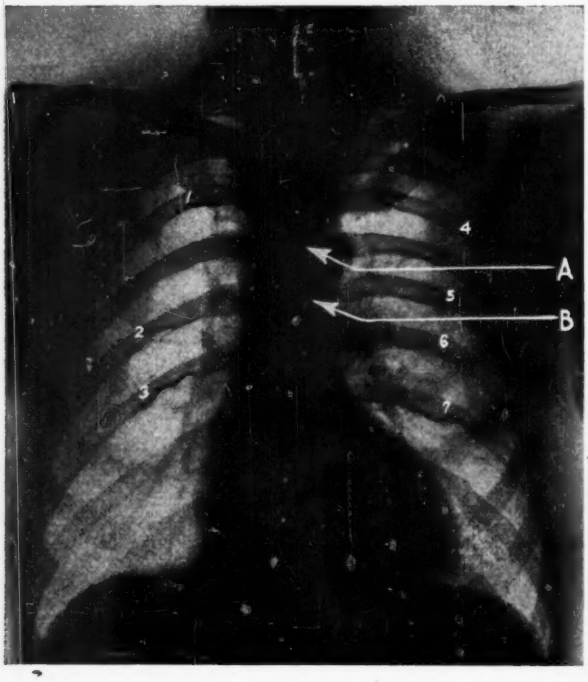

FIG. 1. Roentgenogram of chest showing scalloping of the ribs at 1, 2, 3, 4, 5, 6, 7, 8.
The aortic knob is marked at A. The arrow B indicates the point at which the aortic pulsation appeared to stop at fluoroscopy. The transverse diameter of the heart is just one-half that of the internal diameter of the thorax.

cm. to the left and 2 cm. to the right of the midsternal line. The supracardiac dullness measured 4 cm. The heart was regular and no murmurs were heard.

He was next examined in June, 1934. He felt well, and was working at odd jobs without discomfort. If he walked steadily for some time an ache, which disappeared with rest, would appear in the right calf. Facial pallor was still present. The outlines of the optic discs were clear-cut. The retinal arterioles were narrow and tortuous, and the right infra-nasal artery had a beaded appearance. But slight pulsations were noted on either side of the neck. Over the chest anteriorly, pulsations were visible at the first left and the second right interspaces, and there were vigorous pulsations of the epigastrium. Below the scapulae and over the right lower axilla, pulsating vessels could be felt. The right brachial blood pressure was systolic 166, diastolic 108. The left femoral blood pressure was systolic 130, diastolic 116. The cardiac apex was 9.5 cm., and the left border of cardiac dullness was 12 cm. to the left of the midsternal line. The right border was 2.5 cm. to the right of the mid-sternal line. The supracardiac dullness measured 4 cm. Auscultation revealed an occasional premature beat. In the erect position a systolic murmur was heard at both fourth interspaces, and at the third right interspace, close to the sternum. In the recumbent position a systolic murmur was heard over the second, third, and fourth interspaces on the left and the fourth interspace on the right. The murmur was heard to the cardiac apex on the left and almost to the nipple line on the right. It was heard distinctly over the apex of the epigastrium, and was best heard over the lower left costal cartilage. A systolic murmur was also heard on either side of the four upper dorsal spinous processes, being better heard on the left side. No pulsation could be felt over the abdominal aorta. The femoral pulses were weaker than normal, and the right femoral was definitely delayed there when compared with the right radial pulse. Pulsations of fair strength were felt over the dorsalis pedis arteries, while those of the posterior tibials were feeble.

Fluoroscopy of the chest later by Dr. L. B. Packard showed forcible pulsations of the ascending and transverse aorta. In the region of the isthmus there was a constriction, and no pulsation was visible.

A reproduction of a posterior-anterior roentgenogram of the chest is shown in figure 2. As would be expected from the shorter duration of the process in this patient, the scalloping of the ribs is not so

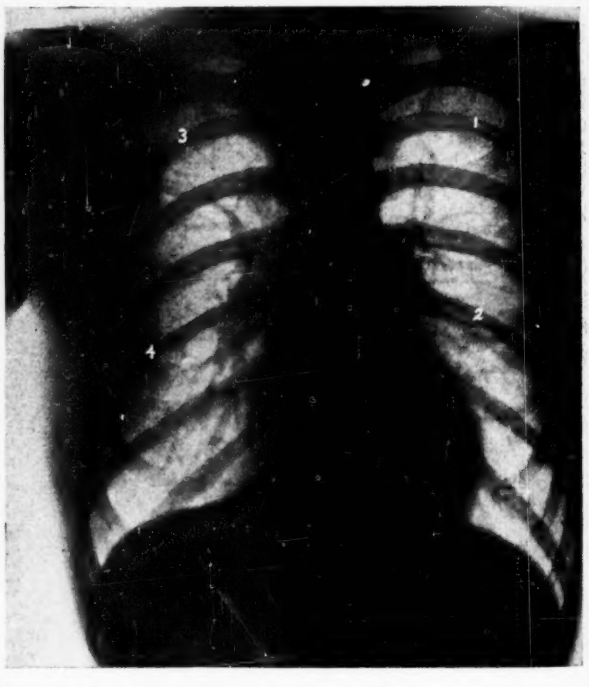

FIG. 2. Roentgenogram of the chest of patient No. 2. Erosions of the ribs are shown at 1, 2, 3, and 4.

deep. For the same reason, perhaps, the aortic knob and left ventricle are less prominent. (The indentations of the ribs have been retouched.)

Because of the interesting findings in two members of this family, the brachial and femoral blood pressures of the other two children and the wife of Albert D. were obtained. Also, the

blood pressures of a sister and the mother of Mrs. Albert D. were determined. The figures obtained, together with the ages of these individuals, are presented in table 2.

TABLE 2

	Mrs. Albert D.	Winthrop D.	Caroline D.	Mother of Mrs. Albert D.	Sister of Mrs. Albert D.
Age	44	16	12	67	48
Right Brachial Blood Pressure	224	126	108	204	184
	130	84	80	120	108
*Left Femoral Blood Pressure	228	152	154	252	240
	200	118	104	216	130

*The femoral blood pressures were taken on the left thigh with the patient prone, and with the chest piece of the stethoscope applied to the popliteal space.

SUMMARY

The occurrence of coarctation of the aorta in a father and son is reported. The mother, the maternal grandmother, and a maternal aunt of the son have hypertension of the usual type.

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VERMONT STATE MEDICAL SOCIETY

CLINICAL REMARKS ON HYPERTENSION AND THE KIDNEY*

THE speaker's remarks were confined to the renal manifestations of primary hypertensive disease (essential hypertension).

It is worthy of emphasis that fatal renal insufficiency develops in only a small proportion of individuals with essential hypertension. Most large series show that well under ten per cent of patients with non-nephritic hypertension succumb to uremia. The younger the hypertensive individual the more apt he is to succumb to renal failure; older hypertensives have a smaller incidence of uremia, and most often terminate with cardiac or cerebral manifestations.

The most generally useful test for estimating the functional efficiency of the kidneys in patients with essential hypertension is the simple specific gravity test. If the patient eliminates a morning urine of specific gravity 1.020 or more after abstaining from drinking after 5 P.M. of the previous day, renal function is adequate at the time and any symptoms present are not due to uremia. In such cases, and they constitute the vast majority of hypertensives, there is no practical necessity for studying the blood chemistry. On the other hand, if the specific gravity test reveals figures under the above-mentioned circumstances, of 1.015 or less, renal function is impaired and the blood chemistry should be studied to determine if nitrogen retention has developed.

The development of renal insufficiency in essential hypertension can often be foretold from the ophthalmoscopic findings. Should the ophthalmoscopic picture include papilledema, it is highly probable that the patient will develop fatal renal insufficiency within two years. This statement holds even though renal function is good and there is little or no albumin in the urine at the time the edema of the papilla is noted. On the other hand, arteriosclerotic lesions of the retina without papilledema convey little or no information regarding the nature

*Résumé of an address by Arthur M. Fishberg, M.D., Associate in Medicine, Mount Sinai Hospital, at the Annual Meeting of the Vermont State Medical Society at Burlington, October 5, 1934. For record and address of author see "This Week's Issue," page 1223.

of the renal process in essential hypertension. In a general way, it may be said that very narrow retinal arteries, especially in the relatively young, convey a poor prognosis in essential hypertension.

Benign paroxysmal hematuria in hypertensive subjects was discussed. This consists in the sudden appearance of macroscopic hematuria, without pain or other subjective accompaniments. The hematuria lasts several days and then clears up. It is presumably of arteriosclerotic or angiospastic origin. I have twice known cases of essential hypertension to be considered as glomerulonephritis because of episodes of such benign hematuria, the existence of which does not seem to be generally known.

The clinical picture of left ventricular failure in essential hypertension was discussed. The cardinal symptom is dyspnea, either exertional or paroxysmal, in the absence of systemic venous engorgement. Cough is a common symptom of left ventricular failure which is often mistakenly attributed to emphysema or bronchitis, and which can often be relieved by digitalization and other measures that improve the circulation. Gallop rhythm and accentuation of the pulmonic second sound are common physical signs in left ventricular failure. Usually, the presence of left ventricular failure is documented by prolongation of the pulmonary circulation time as measured by the injection of saccharin or by other methods. The measurement of the circulation time by injection of saccharin is very simple and can be carried out in the office or the home of the patient. It often detects left ventricular failure in hypertensive patients at a stage when the symptoms are equivocal and the response to therapy excellent.

INDICATIONS FOR AND PRINCIPLES OF SURGICAL THERAPY IN DISORDERS OF THE KIDNEY AND URETER*

BY PAUL W. ASCHNER, M.D.†

THE problems presented in the diagnosis and management of disorders of the kidneys which are of a toxic, degenerative, metabolic or vascular origin are to be discussed by the internists. The most recent addition to methods of investigating the urinary tract, namely excretion urography, is to be explained by Dr. Swick, whose original researches led to the practical application of this procedure.

It is the province of the urologist in disorders of the genito-urinary tract to arrive at an accurate diagnosis which comprises several phases, namely, (1) Anatomical, (2) Functional, (3) Pathological, and (4) Etiological.

(1) The anatomical diagnosis concerns the determination as to whether the urinary tract of the individual is so constructed as to be regarded within the range of normal variations or beyond that range, in which case the exact anomalous conformation must be determined.

(2) The functional diagnosis is concerned first with the total excretory ability of the renal parenchyma in relation to the entire body economy, and, secondly, with the relative or fractional excretory ability of the kidneys, whether they are constituted normally as two separate practically equal units or anomalously constructed in two, three, or four unequal units. The functional diagnosis includes, moreover, the motor and mechanical factors involved in the outflow of the excreted urine from those units. Do they empty normally? If not, what are the factors interfering with normal evacuation?

(3) The pathological diagnosis means determination of the nature of the lesions, their location and extent.

(4) The etiological diagnosis is resolved in several parts. Is the disorder primary in the kidney or secondary to some morbid condition in the outflow tract below it? Is it of congenital metabolic, neoplastic, or inflammatory origin? If bacterial, what organisms are responsible, from what focus have they reached the kidney and by what routes?

Perhaps in no other field of medicine is information so accurate and all-inclusive obtainable as in the genito-urinary tract. It is a function of the general practitioner to be familiar with the protean symptomatology of renal disorders, and to derive as much information as possible by a detailed history of his patient, careful general physical examination and accurate urinalysis. Bacteriological study of the urine, including a search for tubercle bacilli in cases of chronic pyuria, is readily obtainable. Testing the emptying ability of the bladder, that is determining by catheterization the presence or absence of residual urine is a simple procedure, and of great clinical importance. A good roentgenogram of the urinary tract is also easily procured. The method of excretion urography has brought further information within the reach of the well-trained practitioner, but a serious note of warning must be sounded lest patients and doctors be lulled into a false sense of security by deductions based upon films which would not be regarded as satisfactory by the experienced urologist or roentgenologist. It should be emphasized, moreover, that the information obtainable by cystoscopic inspection

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of the bladder cannot be dispensed with in many conditions despite apparently normal excretion urograms.

It is well for the specialist in this field, moreover, to remember that he is a physician first and a urologist second. His armamentarium and surgical technic are but the additional means available in arriving at an accurate diagnosis and indications for therapeutic procedures to restore the patient as nearly as possible to a normal anatomical and functional state under the existing circumstances.

The vague abdominal pains of hydronephrosis, the gastric symptoms of unsuspected renal calculus, the clinical picture of ileus accompanying stone impacted in the ureter, the acute abdominal symptoms of renal infarction, the spinal symptoms of perinephric abscess, the febrile course of silent cortical infection, these are but some examples of the bizarre clinical manifestations of renal pathology, frequently unaccompanied by any changes whatsoever in the voided bladder urine. No patient with abdominal symptoms unexplained by any definite demonstrable organic lesion of the intra-abdominal viscera should be subjected to laparotomy unless a thorough urological survey has given the urinary tract a clean bill of health. Too many harmless appendices, gall bladders, uteri and ovaries have found their way to the laboratory in the past.

On the other hand it may not be amiss to caution the urologist whose training in general medicine and surgery has been neglected or whose experience, limited to his chosen specialty, has caused this general training to fade from his conscious mental processes. Too many diagnoses of ureteral stricture have been and are being made in patients with visceroptosis, spondylitis, ovarian dysfunction, disturbance of the abdominal sympathetic system, metabolic disorders and psychoneuroses. The argument of the successful therapeutic test (in this case, ureteral dilatation) is just as fallacious here as in other fields of medicine.

The further investigation of the urinary tract comprises the following procedures:

(1) Estimation of the total function of the kidneys. By a study of the blood chemistry one may determine whether the balance between catabolism and excretion of the products thereof is being maintained. By use of the phenolsulphonphthalein, urea clearance and urine concentration tests, the excretory ability of the total kidney mass at the time of examination may be estimated.

(2) Estimation of the comparative function of the kidneys. Cystoscopic inspection of the bladder and ureteral orifices in conjunction with intravenous injection of indigo carmine (or phenolsulphonphthalein) and ureteral catheterization give valuable information as to the separate excretory and emptying powers of the separate kidney units. After introduction of the ureteral catheter into the renal pelvis, aspiration will demonstrate the presence or absence of residual urine in the renal pelvis and ureter. A comparison of the urea content, dye concentration, microscopic findings and bacteriological study of the separate ureteral specimens gives data of great importance in arriving at accurate diagnosis.

(3) Use of the waxed tip bougie to detect calculi which are not opaque to the x-ray.

(4) Roentgen examination with opaque catheters in situ to determine the course of ureters, and relationship to shadows suggestive of calculi.

(5) Retrograde pyelography and ureterography.

(6) Pyeloscopy and serial pyelography.

(7) Cystogram.

(8) Intravenous or excretion urography.

By a series of some sixty lantern slides selected from my collection of about six hundred, the diagnostic features of the more common and important disorders of the kidney and ureters are herewith demonstrated. They may be classified as follows:

Congenital anomalies such as single kidney, bifid pelvis, bifid ureter, double kidney and ureter on one or both sides, hypoplasia, ectopia, crossed ectopia, horseshoe and fused kidneys.

Acquired malpositions.

Hydronephrosis and hydroureter of various etiologies.

Pyelonephrosis and pyonephrosis.

Cortical infection, carbuncle, perinephritic abscess.

Renal and ureteral tuberculosis.

Renal and ureteral calculi.

Ureteral strictures, ureteritis cystica, ureterocele.

Solitary and multiple cysts of the kidney.

Polycystic kidney.

Neoplasms of the kidney and renal pelvis.

In connection with this lantern slide demonstration I wish to discuss the indications for and principles of therapy which may be summarized as follows:

(1) Treatment of renal infections without obstruction to the outflow system.

Perinephric suppuration whether of coccal or bacillary origin requires simple incision and drainage. Metastatic staphylococcus cortical infection whether unilateral or bilateral occasionally resolves without suppuration or may rarely discharge itself into the calyceal system. Cortical abscess and carbuncle of the kidney may require simple drainage, local excision, or partial nephrectomy. In the rare diffuse cortical infection by the staphylococcus or streptococcus and when renal vein thrombophlebitis is suspected (even in bacillary types of infection) nephrectomy becomes necessary.

Bacillary infections are usually bilateral and involve the pelvis and ureter. They tend to spontaneous healing in the acute stage when aided by forcing fluids and alkalies, or urinary antiseptics of the azo dye variety. Ureteral catheterization and indwelling ureteral catheters frequently relieve the virulent persistent case. In occasional desperate instances bilateral decapsulation with or without nephrostomy may be necessary. Nephrectomy in this type of case is to be reserved for the rare strictly unilateral fulminating case, particularly if thombophlebitis of the renal vein is suspected.

(2) Treatment of obstructive lesions.

The management of unilateral obstruction of the ureters is influenced by the pathological lesion, the location of that lesion, the presence or absence of infection and the nature and severity of such infection. If the obstruction can be overcome by ureteral catheterization, immediate relief of pain and control of infection may be accomplished with preservation or improvement of excretory function, by virtue of reëstablished drainage. A calculus lodged at the ureteropelvic junction may be dislodged back into the pelvis, or a calculus in the lower ureter may be passed by the catheter and external drainage accomplished by fastening the catheter in place. Not infrequently such drainage for forty-eight hours may be followed, after removal of the catheter, by spontaneous passage of the stone. In any event the method affords more prompt relief of renal colic than the use of narcotics. Occasionally a calculus may be extracted from the lower ureter by instrumental means. If impacted in the intramural portion, incision or fulguration of the orifice may induce passage.

In the presence of infection, maintenance of drainage is carried out by pelvic lavage through the indwelling catheter with sterile saline, boric acid, or acriflavine solution 1 to 4000. If the obstruction cannot be overcome, or if systemic and local signs of infection persist with evidence of severe pyelonephritis, despite catheter drainage and pelvic lavage, external drainage by means of nephrostomy plus decapsulation is the procedure of choice. The immediate problem here is the renal damage caused by blockade and infection. With relief of these factors the ureteritis subsides, and not infrequently the calculus passes without colic per vias naturales despite continuous nephrostomy drainage. If the stone does not pass it is a simple procedure to remove it by ureterotomy when the renal infection has subsided.

In the management of ureteral calculi it is important to remember that probably eighty-five per cent of such stones pass spontaneously or with instrumental assistance. Intravenous urography is of great aid in determining the anatomical and functional status in these cases. Impairment of function and infection are the dangers, and meddlesome manipulation may add to them. Removal by ureterotomy must be resorted to in obstinate cases before too much damage has been done, normal internal drainage being restored by removing the stone. The ureter must be sutured carefully so as not to constrict it, drainage by rubber dam down to the suture line is imperative.

Nonspecific strictures of the ureter, and postoperative strictures are relatively uncommon lesions. In the lower ureter gradual and progressive instrumental dilatation is effective. In the upper ureter, surgical exposure of the affected area and retrograde dilatation may be necessary. In rare instances resection of the diseased segment and anastomosis have been resorted to. Occlusion of the lower ureter by accidental ligation during hysterectomy or other pelvic operations may necessitate temporary nephrostomy and subsequent ureterovesical neostomy.

Obstructions caused by aberrant vessels crossing the ureteropelvic junction require either division of the offending vessels or, if they are large and supply large segments of kidney tissue, division of the ureter and reimplantation into the renal pelvis to circumvent the impediment.

Interference with renal drainage due to ptosis or other acquired malposition is cared for by nephropexy.

Hydronephrosis due to stenosis at the ureteropelvic junction, congenital valve formation or S-trap insertion requires plastic procedures of various types, it being of utmost importance to protect the kidney by nephrostomy and the ureteral plastic by an internal splint in the form of a catheter brought out through the renal pelvis and the nephrotomy incision.

(3) Renal Calculi.

A calculus a centimeter or less in diameter in an apparently normal kidney pelvis without evidence of infection may well be left alone. If the calculus is incidental to stasis in the kidney, the condition causing such stasis should be remedied prior to or simultaneously with its removal. The choice of procedure in surgical removal of renal calculi is by pyelotomy, for this does not damage renal parenchyma. If multiple stones are present, some of them lodged in calyces, it may be possible to extract them with properly constructed forceps. My own experience, however, has proved that the ungloved little finger is a superior and safer means of locating and dislodging calyceal calculi. In other instances small nephrotomy incisions are made with a finger supporting the stones through the pyelotomy wound, as each one is cut down upon and extracted. In the presence of infection it may be advisable to introduce a small tube or Pezzer catheter into the renal pelvis by way of one of these small nephrotomy wounds. The pyelotomy incision is then

closed with interrupted sutures, reinforced by perirenal fat.

In cases of stag horn calculi and other varieties of multiple calculi, I have practically abandoned the procedure of splitting the kidney along its convexity as it entails too great risk of hemorrhage, infection and necrosis following the necessary suture repair. Adequate exposure is obtainable by liberal pyelotomy incision and extension of this along the posterior surface of the kidney transversely, permitting approach to the various calyces. An elastic ligature placed temporarily about the vascular pedicle permits unhurried and complete removal of stones. The repair is very simple, a few approximating sutures suffice. Release of the ligature and a few minutes of manual compression control all bleeding. A rubber dam placed about the organ insures adequate drainage.

In the management of multiple stones the control by x-ray on the operating table is an essential safeguard for a complete removal of fragments.

Partial nephrectomy is indicated when calculi are confined to a lower or upper calyceal sac formed by cicatricial stenosis of the pelvi-calyceal junction. In congenital double kidney with stones and infection of one segment, heminephrectomy may be the procedure of choice if blood supply is favorable.

(4) Complex calculus problems.

In the management of bilateral ureteral calculus with suppression and impending uremia, if one ureter can be passed and the kidney drained by catheter, the opposite impacted stone should be removed surgically. If both sides can be passed by catheter, the infected side should be operated upon first.

Given a ureteral calculus on one side and a renal caculus on the other, passage or removal of the ureteral stone is preferable as the first step.

With calculi in both kidneys the infected side should be operated upon first. The simpler surgical problem should be attempted first whenever possible, for example pyelolithotomy on one side should precede pyelonephrolithotomy on the other. The infected side or the one with more impairment of function should be attacked first, other things being equal.

NEPHRECTOMY

Nephrectomy in unilateral calculus disease or hydronephrosis should be carried out only when necessitated by infection or extensive destruction of parenchyma by back pressure. It is not an infrequent experience for a conserved kidney to be the means of supporting life at some subsequent time when the formerly healthy partner becomes the seat of disease necessitating its removal.

Pyonephrosis is often best managed in two stages, nephrostomy to control infection and nephrectomy secondarily performed for permanent cure. Subcapsular removal is usually necessary in these cases and the use of pedicle clamps left in situ is often a wise technical choice in difficult cases.

In the removal of the kidney for tuberculosis the perirenal fat should be included with the specimen, as much of the ureter being removed as possible, the stump being ligated, carbolized and dropped back. Complete hemostasis and closure of the wound without drainage appears the best safeguard against tuberculous wound infection and fistula.

In the removal of renal tumors the perirenal fat should also be included. The lumbar route is adequate in smaller lesions, the transperitoneal route in the large ones. By the latter approach the pedicle may be exposed and secured as the first step in the procedure.

In tuberculous stricture of the ureter, and in the presence of ureteral tumors ureteronephrectomy is indicated. The lower ureter is exposed through a median hypogastric or right pararectus incision, divided between ligatures at the ureterovesical junction and then mobilized upward. The patient is then turned on the opposite side and the kidney exposed and removed by the lumbar route with the ureter attached. In some instances of ureteral tumor at the orifices, partial cystectomy may be carried out with this procedure.

In conclusion, the objective of the urologist in renal and ureteral disease is conservation in benign lesions and radical removal when life is at stake.

MISCELLANY

VERMONT DEPARTMENT OF PUBLIC HEALTH

NOVEMBER, 1934

The incidence of communicable diseases reported during the month of December to this Department is as follows:

Chicken pox 259, diphtheria 11, measles 10, mumps 14, scarlet fever 64, typhoid fever 8, undulant fever 1, tuberculosis 6 and whooping cough 143.

Laboratory of Hygiene made a total of 1,963 examinations in November. A list of the examinations follows:

Examinations for	diphtheria bacilli	405
"	" Widal reaction of typhoid fever	41
"	" undulant fever	43
"	" gonococci in pus	150
"	" tubercle bacilli	158
"	" syphilis	537
"	of water, chemical and bacteriological	63
"	" water, bacteriological	193
"	" milk, market	184

Examinations of milk submitted for chemical only	8
" " milk submitted for microscopical only	79
" " foods	2
" " drugs	0
" for courts, autopsies	9
" " courts, miscellaneous	36
Evidence for rabies (animal heads)	0
Miscellaneous examinations	54
Autopsies to complete death returns	1

Twenty-nine cases of gonorrhea and twenty-three cases of syphilis were reported to the Division of Venereal Diseases. Wassermann outfits numbering 986 and gonorrheal slides numbering 352 were distributed to doctors this month.

The director of the Division of Tuberculosis gave seventeen talks to groups of teachers, fifteen talks to school children and nine talks to other groups.

The nurses of the Infantile Paralysis After-Care made 214 home visits on patients, visited four physicians, and seventeen social service calls. Two patients were admitted to the Massachusetts General Hospital. Ten pieces of apparatus have been repaired and twenty-seven orthopedic corrections made to shoes. Sales during the month amounting to $95.24 were reported by the Vocational Worker of this Division.

The State Advisory Nurse has devoted practically all her time this month to organizing the work of the coming project under the Vermont Emergency Relief Administration to be carried on this winter for unemployed nurses.

MEDICAL PROGRESS

PROGRESS IN DERMATOLOGY 1934

BY HARVEY P. TOWLE, M.D.,* AND J. L. GRUND, M.D.*

IN reviewing the progress of dermatology as shown in the literature for the past year a new trend became evident. Whereas for years past the laboratory has dominated the literary picture, during this past year the clinic has attained increased prominence. The number of papers dealing with therapy, the relief and cure of disease, has grown most perceptibly. The articles are not limited to one particular field but cover a large number of diseases as will be seen from the selections given below.

A somewhat startling example of this is to be found in the application of the newly found facts concerning allergy in the treatment of fungus disease. Kerr, Pascher and Sulzberger[1] have used extracts of Monilia and Trichophyton in the treatment of eczematous ringworm. They begin the report of their observations of this new method by calling attention to the fact that the monilia group of fungi are very common inhabitants of the skin. They may be present in the toe nails. the mouth, the gastro-intestinal tract, and are commonly present in the intertrigenous and other moist areas. They are powerful sensitizers, resembling in this respect the trichophyton interdigitale. The sensitization moreover is specific. Neither the monilia group nor the trichophyton sensitizes to the other group but only to its own.

The authors studied a group of twelve cases of dermatophytosis with dermatophytides. First, they determined by means of intradermal injections of extracts of the monilia group, on the one hand, and of the trichophyton group, on the other hand, the proportionate influence which each group had in the production of the disease, using the degree of reaction to the respective groups as a yardstick. Treatment was then based upon the calculated results.

They found that, of the twelve cases studied, eight were hypersensitive to both trichophyton and oidiomycin and that four were sensitive to trichophyton only. From these results they concluded that both organisms are present in many cases of hand and foot eruptions and, further, that it is almost impossible to determine which group is the major offender. They also found in these cases of double sensitization that they could not desensitize the patient against both groups but only against one. This but emphasized the fact that the two groups of fungi are immunologically different. Nevertheless, the authors are convinced that the attempt at a combined desensitization to monilia and trichophyton is so logical that they have adopted it as a routine measure in the treatment of persistent fungi cases. The success which has followed has, in their belief, justified the conviction.

This experiment confirms a similar one reported a month earlier by Ruete and Scholz[2]. These two writers studied fifteen patients. Every patient with epidermophytosis responded positively to a test made with a filtrate from epidermophyton cultures. The test was negative in a trichophyton infection and in a case of tinea versicolor which proves to the authors that the epidermophyton test is specific. It proves to them also that epidermatophytosis is more than a local disease. Because of their belief that epidermatophytosis is more than a local disease

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and because of their belief that epidermophytin is specific for epidermophytosis, they were led to try out epidermophytin as a therapeutic agent. They report that every one of the fifteen patients was cured promptly. The injection was not painful and produced no secondary effects beyond a slight itching. But, they also used iodine and a sulphonated bitumen which rather throws doubt upon their conclusions.

We have known for years that it is possible to introduce certain therapeutic substances through the skin by friction. For instance, one of the oldest methods of treating syphilis has been by rubbing mercurial ointment into various portions of the body. But the study of such methods has been largely by the trial and error method. If one incorporated a certain drug in a certain vehicle and by rubbing the preparation into the skin got certain effects which could be attributed to the drug it was concluded that the drug had been carried through the skin. Reitz[3] in applying the inunction method of treatment to leprosy, approached the study from a more precise angle. He conceived a theory from his research work that only substances which are compatible with the cholesterol of the skin and which are perfectly dissolved in the vehicle have any chance of getting through the skin and being absorbed. As a corollary to this theory he adopted a second to the effect that the skin is the principal and primary seat of leprosy because the bacteria are imbedded either in the cutis or in the subcutaneous tissue. He put these theories to the test in the San Lazaro Hospital. He used no fixed formulae but fitted each one to the needs and special indications of each case, seeing to it always that each remedy used was compatible with the cholesterol in the skin and was perfectly dissolved in the vehicle. He reports the most gratifying results. His technic was to treat the whole body daily. First he gave a soap and water bath. Then the selected remedy was rubbed in over the whole body. The front was cared for by the patient himself. The back was cared for by another patient. Reitz reports that the lesions changed radically in a very short time. Infiltrations of every character and kind disappeared leaving only a faint stain which, too, disappeared in a short time. The skin, once the seat of frightful lesions, became clean and smooth. Most striking of all is Reitz's statement that the skin now showed no signs of the disease and the blood examination revealed no bacilli.

If Reitz can obtain such results in leprosy it would seem that it might be worth while to study his inunction method in other diseases such as acne and furunculosis.

Another example of putting laboratory findings into clinical practice is furnished by Burgess in his studies of autohemotherapy[4]. The method is not new, but his conception of its action is. Burgess' argument runs in this wise. Only about sixteen per cent of normal human beings are sensitive to their own proteose, whereas in a group of patients afflicted with such widely different diseases as psoriasis, eczema, varicose dermatitis with sensitization rash, chronic urticaria, toxic erythema, furunculosis, Besnier's prurigo and idiopathic generalized pruritus, he found that sixty-seven per cent were sensitized, that is, gave positive reactions to proteose. The blood of these patients contains an antigen so that, when their own blood is injected, it desensitizes them. His results, he says, confirm the theory. He reports that all but one case of acute psoriasis were greatly improved within two months. Chronic psoriasis, however, was unaffected. The effect in cases of varicose dermatitis, which had developed a generalized dermatitis, was particularly striking. Certain eczemas were favorably affected, even to the cure of some. Good results, too, were obtained in urticaria. Burgess calls attention to the fact that when the autochemotherapy was successful in curing the disease the patient had become insensitive to the proteose test. Patients who were not relieved had either had a negative proteose reaction before treatment was begun or had remained negative in spite of much treatment.

In a communication to the *Journal of the American Medical Association,* Guy and Jacob[5] relate their impressive success in the prevention of impetigo neonatorum. The method is extremely simple but, according to the statistics, very successful. They cause the baby to be cleansed thoroughly with sterile liquid petrolatum as soon as possible after birth. Before it leaves the delivery room the baby is anointed from head to foot with a two per cent ointment of ammoniated mercury. In the nursery the daily cleansing is accomplished with sterile cottonseed oil. The buttocks and arms are cleansed with cotton balls and cottonseed oil. Soap, water or powder is not used. As a result of this routine procedure, they report these statistics. In 1929 among 2344 births there were thirty-four cases of impetigo, twenty of which occurred in one month. In 1930, when the routine described was instituted, there were but two cases of impetigo. In 1931 there were 2884 births and but one case of impetigo; in 1932, 2522 births and one case; and in 1933 no cases.

Every dermatologist has met cases of recurrent herpes simplex which simply will recur in spite of all measures taken to prevent it. If other investigators take Hruszek's finding, that will not happen in the future. Hruszek[6] has treated one hundred cases of recurrent herpes simplex by inoculating the arm with the contents of the herpetic vesicles. The inoculation is repeated every second day until it no longer "takes". But one of the one hundred cases recurred after treatment by this method.

Warts are always a perennial topic of discus-

sion. Cure after cure has been reported only to be denied. When Bruno Bloch claimed in 1927 that he could get as good results by suggestion as his colleagues could by their injections and ointments and washes, his claim was ridiculed at first. But as time went on the admission grew more and more frequent that perhaps he was right. Allington[7] is one of the latest to put Bloch's claim to the test. One hundred and five patients with various forms of warts were given intramuscular injections of 0.1 to 0.3 Gm. of sulpharsphenamine. One hundred and twenty patients were given similar injections, under conditions strictly similar in every respect, of distilled water colored to match the sulpharsphenamine solution. They were able to follow up eighty of the sulpharsphenamine patients and eighty-four of the placebo patients. The great majority of both classes received but one injection. The results were almost alike.

In 1932 Dr. Sophie Lurie reported one hundred per cent of cures in thirty-four cases of warts treated by injections of bismuth salicylate. Lunsford, Thomson, Binkley and D. S. Fox[8] followed her technic in sixty-one cases. Their conclusion was that "the 50% of cure in eight cases in children and the 28.2% of cure in thirty-nine cases of adults would indicate that bismuth therapy has a limited place in the treatment of verruca vulgaris."

We have always with us those who have not enough hair, on the one hand, and those who have too much hair, on the other. It would seem from the London Letter to the *Journal of the American Medical Association*[9] and from an article in the Medizinische Klinik[10] that our recently acquired knowledge of the endocrines and of the hormones is being invoked for the relief of these opposed conditions.

The London Correspondent[9] writes of a report made to the Section of Dermatology, Royal Society of Medicine, by Dr. A. D. K. Peters of a new treatment of hirsuties in the female based on recent advances in our knowledge of the sexual hormones. A woman of thirty-eight complained of a heavy growth of hair on the lip, the bearded face, the thighs and the legs. She asked for its removal. Dr. Peters gave her crystalline trihydroxyestrin by mouth, one hundred Doisy rat units, daily for a week. Nineteen days later, he reports, the hairs of the moustache and beard areas began to fall. This continued for a while and then gradually ceased. The treatment was repeated and again the hair began to fall. Bare, oval areas about a half inch long appeared on the chin. A month later crude keto-hydroxyestrin in a three times greater dosage (303 Doisy units) was given orally every day for ten days. Two or three hairs fell every day. The dose was doubled but the fall was not increased. Peters called attention to the observation that the fall was always greater from the chin than from the lip and each course of treatment was followed by an earlier but diminished response. In the end there remained a moderately heavy moustache but only a few stiff hairs on the chin. But the hair was beginning to grow again.

We know that there is a relationship between the ovaries and the pituitary glands. It is, therefore, particularly interesting to note that whereas Peters tried to cause a loss of hair by the administration of trihydroxyestrin, Höcker[10] tried to cause hair to grow by means of pituitary medication. Höcker treated a woman of nineteen who had first developed small hairless areas on the occiput and finally lost all her hair. The woman had always been subject to menstrual disturbances, delayed menses and for a time amenorrhea. She also exhibited symptoms of hypofunction of the anterior pituitary lobe. Under the oral administration of a follicular hormone a colorless, downy growth of hair appeared and menstruation became normal. In three months the growth of hair stopped. The dose of the hormone was increased. Now 350 mouse units were given by mouth and 200 by subcutaneous injection. Gradually the hair growth increased again but, three months after medication was stopped, the hair began to fall out again. The daily administration of 1000 mouse units by injection again stimulated a growth of hair. The patient was under observation for three years but the improvement never went on to complete cure. Höcker's conclusion is that hormone therapy alone is not sufficient to reëstablish normal hair growth.

Several points of similarity will be noticed in Peters' and Höcker's reports. Both reported acceleration of effect with the renewal of medication, the gradual loss of effect, the necessity of increased dosage with the passage of time and final failure to obtain complete cure.

From England still come enthusiastic reports on the treatment of lupus erythematosus with gold compounds. One of the latest is by J. L. Franklin[11] who used Sanocrysin, the double thiosulphate of gold and sodium, in thirty-one cases. Twenty were "cured", seven were improved, four were resistant. His best results were obtained in patients who had had the disease for relatively short times, five years or less. The type of the disease was also an important factor in that the results were best in the superficial types. He believes that, once healed, the patient will remain "cured" provided that the treatment was adequate. He believes that relapse means insufficient primary treatment.

Franklin's exposition of his theory as to the action of sanocrysin is interesting. In the first place, he believes that lupus erythematosus is the cutaneous response to a bacterial toxin, not necessarily to one specific toxin but to any of a variety. He feels that sanocrysin acts upon the bacterial organism as salvarsan does upon the organism of syphilis. When sanocrysin is in-

troduced into the blood stream the gold containing ion unites with some tissue protein to form a substance which has a direct lethal effect upon a variety of organisms. He thus explains its effect in lupus erythematosus. He believes that this theory affords a practical explanation of the similarity between the untoward effects of gold therapy and the accidents occurring with salvarsan.

Less frequent reports are received of the use of gold salts in the treatment of vitiligo. Burgess[12] reports a case with a very successful outcome. Pigmentation of the leukodermic areas was noted within three weeks of the first administration of oil of bergamot and the ultraviolet rays externally and of sanocrysin intravenously. Ten weeks later pigmentation was complete. There has been no recurrence.

Whitfield[13] has published some interesting observations on acne. He believes that in intelligent hands the Roentgen ray can cure any case of acne vulgaris. But after roentgenization the patient must not be exposed to either the sun or to the ultraviolet ray. As to the ultraviolet light, he feels that it has no value. If there is improvement with its use, it is only temporary. Diet, he believes, plays a definite part in the successful management of acne. Internally, he has found sulphur a help. Externally, he advises compresses of weak bichloride solutions.

Since the use of deep Roentgen ray over the spinal sympathetic ganglia in the treatment of diseases of the vasomotor system and in such diseases as lichen planus there has been manifest a disposition to modify the x-ray technic in a variety of other diseases. Rieva Rosh[14] has experimented with this method of raying the sympathetic ganglia in psoriasis. She subjected forty-six cases of generalized psoriasis to high voltage x-rays applied over the spine at the levels corresponding to the nerve supply of the affected regions of the skin. The usual dose was 860 R or a seventy-five per cent erythema dose. The effect was not immediate but was observable in from three to six months after treatment. In a very few obstinate cases the effect was delayed for as long as a year. It is reported that every case, without exception, cleared up.

The interesting fact about the newer methods of treating the scalp is the use of the fluorescent lamp not only to make the diagnosis but also to determine the progress of treatment. Davidson and his colleagues[15] treated thirty-eight patients having ringworm of the scalp with thallium acetate. Seven cases had no clinical signs of ringworm, yet were adjudged infected because they showed green fluorescent hairs under the Wood's light. The patients were always clinically cured before they ceased to show fluorescent hairs under the fluorescence lamp.

All roentgenologists are agreed that early treatment of cancer of the lip favors permanent cure. They differ more or less as to how irradiation should be given. Pfahler and Vastine[16] have recently published their technic. If there are no palpable nodes they surround the cancer with a line of local destruction by electrodesiccation after local anesthesia. They then destroy the main growth by the same method. After electrodesiccation they use a cross-fire irradiation with high voltage Roentgen rays filtered through 0.5 mm. copper. Irradiation is repeated up to a total dosage of 1400 Roentgens (Pfahler's method). Where irradiation is not practicable they employ the radium element both interstitially and on the surface. When employed interstitially the radium is filtered through 0.3 to 0.5 mm. of platinum and when on the surface through 0.2 mm. of platinum. In addition high voltage Roentgen treatment is given to the regional lymph nodes.

While many articles on allergy continue to appear, most of them are clinical studies and show little that is new. A certain number of writers, however, show a disposition to discriminate in accepting all tests for allergy at their face value. For example, Mendelsohn[17] found that in 262 cases of eczema, dermatitis venenata, prurigo, neurodermatitis, and urticaria, intradermal tests were of little value in determining the cause of the eruption.

Walzer and Grolnick[18] question the value of the skin tests in papular urticaria and in prurigo. They report a study of thirty-six cases of papular urticaria and five of prurigo mitis. Of the thirty-six cases of urticaria they found that forty-seven per cent gave a positive family history of atopy and that about one-third of these (6) gave a past history (asthma and infantile eczema). Of the series of thirty-six cases only such as gave positive family history of atopy (forty-seven per cent) gave a positive reaction to one or more allergens. The remaining fifty-three per cent were negative. The same rule obtained in the cases of prurigo. Only such cases as had a positive family history of atopy gave positive skin tests. From these facts, Walzer and Grolnick feel that the positive skin tests encountered in these diseases bear no relation to the etiology of the disease itself but only to the atopic conditions associated with them. (Forty years ago they called this inherited condition a dyscrasia.) Specific therapy based upon the positive tests obtained was found to have no value. Their conclusion is that, while papular urticaria and prurigo are atopic diseases, in them skin tests have neither diagnostic importance nor value as a guide to treatment.

The search for an exact definition and understanding of allergy still goes on. The general assumption has been that the presence of eosin-

ophiles in numbers in wheals is an evidence of allergy although this is disputed by a few. In our 1933 report we noted that Werner Jadassohn disputed the assumption on the ground that with such non-allergic substances as atropin and histamine he could reproduce a pathological picture indistinguishable from that produced by allergic substances. Knott and Pearson[19] have conducted a research study to prove or disprove the theory that it was possible to distinguish between an allergic and non-allergic reaction by examination of the mixture of wheal fluid and the blood obtained by pricking the site of a histamine wheal. The authors found that the eosinophil concentration is doubled in the wheals produced by the injection of appropriate proteins into sensitized patients and that when proteins to which they were not sensitive were injected into the same persons there was not an eosinophil concentration to the same degree. Although it is true that some eosinophil concentration occurs in the histamine wheals produced in normal controls the authors conclude that the concentration is more pronounced in patients presenting allergic tendencies. The difference, therefore, seems to be one of degree and not of kind.

Sulzberger and Feit[20] present another phase of the allergy problem. Their observations were made on a series of twenty children, with heart disease, most of them below eleven years of age. Quantitative injections of Old Tuberculin were given. It was found that a marked anergy was present which was in striking contrast to the high percentage of positive tests usually obtained in urban groups of the same age. They think that the difference in the two groups may be explained logically by the presence of cardiovascular disease in the one and its absence in the second. Somehow the cardiovascular disease determines what degree of allergy it is possible to excite in such patients.

Tedstrom's case[21] is interesting as showing that an antigen circulating in the blood in one person may be transmitted to another and still remain active. A blood transfusion was required by a patient who previously had been free from allergic manifestations. His donor had urticaria. After the transfusion the recipient developed urticaria. Tedstrom believes it is explainable by the transmission from the urticarial donor to the urticaria-free recipient of the active substance causing the urticaria, probably, Tedstrom thinks, the histamine-like substance of Lewis.

The relationship of erythema nodosum to tuberculosis is still a matter of fierce debate. Göbel and Schuhardt[22] support the claim of a positive relationship in a recent argumentative article. Those who deny the relationship, say Göbel and Schuhardt, protest that the incidence of erythema nodosum is so low compared with the incidence of tuberculosis as to lack significance. To this the writers reply that tuberculosis is but one factor in the production of erythema nodosum, that there are other factors also concerned. The tuberculosis which excites the nodosum may be a primary infection or superimposed, but it may also be an old process which has apparently run its course. The factor necessary to the development of the nodosum may be (1) a constitutionally weak defense; (2) an acquired endogenous disturbance in the defense mechanism, such as a disturbance in the hormonic cycle or the seasonal increase of reactivity; (3) an exogenous disturbance in the defense powers, such as an infectious disease or the application of tuberculin. They report a case to illustrate their argument. A boy was admitted to the Institute. The examination at the time of admittance demonstrated that he was already infected, for tubercle bacilli were found and his reaction to tuberculin was severe. He was also proved to have an intestinal infection (lambliasis) which produced a negative anergy. When he was put with other tuberculous patients he developed a new infection which, because it occurred at a time of negative anergy, led to erythema nodosum and also produced a tuberculous infiltrate with typical roentgenologic and clinical aspects.

Aguirre[23] presents as proof of the tuberculous nature of erythema nodosum the record of a guinea pig infection by material derived from a case of erythema nodosum. A three year old child developed erythema nodosum in the left tibial region. Material excised from the region was inoculated into guinea pigs. A generalized tuberculous reaction followed.

Rotnes[24], too, believes in the tuberculous origin of erythema nodosum especially in children. In adults, he says, the evidence is not so clear. He had four cases in sisters, two of whom were adults and two were children. None were tuberculous before. A husband of one of the sisters lived with them during a period of active tuberculosis which subsequently led to his death. During his illness and shortly before his death all of the sisters developed erythema nodosum. All became positive to the von Pirquet test. In the children, x-ray examination showed well-marked signs of tuberculosis in the lungs. In the adults there were no signs of tuberculosis. Rotnes feels that the tuberculous man was the source of the erythema nodosum infection.

These cases remind one of a similar observation by Begg recorded in the last Report in which the patients were exposed to active tuberculosis and developed erythema nodosum. Begg considered the skin disease to be an allergic manifestation of a recent tuberculous infection.

Von Moritz[25] also would consider erythema nodosum an allergic manifestation of tuberculosis. He cites the cases of three children who were admitted to the hospital for various rea-

sons. At the time of admittance none of the children could be demonstrated to be tuberculous. The physical examination, the tuberculin tests and the history were negative in all. Yet five or six weeks after admission all three children gave tuberculin positive tests and all developed erythema nodosum. Von Moritz is convinced that the reason that they had erythema nodosum was because they had become tuberculous and that the appearance of the erythema nodosum marked the end of the incubation period of the tuberculosis.

Bruusgaard[26] writes of a "primary complex" in tuberculosis whose complete picture is most often seen in children. In patients, previously free from tuberculosis, there appears in the skin a primary infection with a sore and a subsequent definite and often pronounced swelling of the regional lymph glands. The marked lymphangitis which usually accompanies this process denotes the spread of the bacilli to the regional nodes. The sore marks the port of entry of the bacilli. He offers three cases in children to illustrate. In one child a sore developed at the left angle of the mouth. The submaxillary glands swelled and the glands along the sternocleido muscle. The von Pirquet test became strongly positive and miliary tubercles and tubercle bacilli were found on histologic examination. A second child showed a sore on the right leg with lymphangitis and enlarged inguinal glands. A third had a primary tubercular sore on the right arm accompanied by lymphangitis and swelling and softening of the regional glands. These are the pictures of the "primary complex" in tuberculosis which is found only in those not previously infected.

Rauschkolb[27] has made an interesting statistical report on sixty-eight cases of tuberculosis of the skin. He found that the cases could be separated into two groups, one composed of cases of external origin and one composed of cases of internal, hematogenous origin. He observed further that the white patients were more apt to have infections from without and the Negroes infections from within. Thirty-seven per cent of the total cases occurred in the first year of life and ninety per cent of these were of hematogenous origin. Sixty-one per cent had demonstrable pulmonary tuberculosis. More than fifty per cent of the cases with lichen scrofulosorum and twenty-five per cent of the cases of papulonecrotic tuberculosis had phlyctenular keratitis. Twenty-five per cent of the sixty-eight patients died of pulmonary tuberculosis. Fifty per cent were healed or improved, twenty-five per cent were not improved. Cases of lupus vulgaris and of tuberculosis verrucosa cutis did well when curetted and the wound powdered with potassium permanganate.

Hollander and Schlesinger[28] have published an interesting thesis on the nature of the sarcoid of Boeck. As a result of their studies of the blood, the writers came to the conclusion that sarcoid is tuberculous in nature, that it is a distinct variety of tuberculosis, and is the product of an attenuated strain of the tubercle bacillus occurring in persons whose reactions differ from the average. In two cases of sarcoid, in one of tuberculosis of the hypoderm and in one case of lupus pernio, the blood showed systemic disturbance. The picture in lupus pernio is not constant but varies from a leucocytosis, as in sarcoid, to a leucopenia, as in tuberculosis of the hypoderm. In all the conditions, the type of reaction depends upon certain factors, such as the type of tubercle bacilli, the species and number, on the ability of the host to cope with the infectious agent, that is, on his resistance, or in a lack of immunity, on the age of the process and, not improbably, on climatological conditions.

Sulzberger[29] discussed the problem of sarcoid more from the allergy angle. He accepts the etiological rôle of the tubercle bacillus. In the case of sarcoid which he reports both skin and lungs were involved, the x-ray examination of the lungs revealing a picture resembling that of a miliary tuberculosis. It has been noted that in sarcoid the tuberculin test is usually negative. This, Sulzberger explains, is due to the presence of a state of anergy. Even high concentrations elicit no response which is remarkable when compared with the high percentage of positive tests yielded by average people. He reports a good result in his case from a diet poor in salt, high in vitamins and containing potassium and calcium.

Since the advent of the Workmen's Compensation Laws, industrial dermatology has become of constantly growing importance. Consequently, it is no surprise to find many articles on the subject. The diversity and extent of the field which industrial dermatology covers already are most amazing and as will be seen by the selected references given below the field is constantly being extended.

Not many years ago a papulopustular eruption among coal miners would have excited only a passing interest but today its compensability gives it new importance. Therefore, Dowling and Brain[30] have found it worth while to discuss such an affection at considerable length. The course and symptoms of the disease were as follows: The afflicted workman would at first complain of an irritation of the skin. Within a few hours a widespread eruption would appear involving the extensor aspect of the thighs, knees, legs, buttocks, and forearms, and in many cases the inner aspect of the forearms as well, the waist line, the chest, and the back. This course and distribution were constant in every case. Even though the man ceased to work, the eruption continued to spread during the next few

days but then gradually died down. If the man returned to work too soon the eruption promptly relapsed. Occasionally one who seemed to have recovered would relapse upon his return to work. The eruption itself was composed of closely set, punctate lesions usually thickest on the hairy parts. The lesions were discrete and papular. Some were almost flat. Many were scaly. A certain number were capped by small crusts or pustules. In patients with dry skins, the eruption consisted of flat, dry, scaly patches of an eczematous type. On the non-hairy regions the papules were suggestive of those seen in a seborrheic eczema. A biopsy of one of the papules, examined serially, showed a superficial pustule with a subcorneal colony of organisms. Smears and cultures from the pustules revealed the staphylococcus albus and occasionally the aureus. The reporters write that there are three possible solutions of the cause. (1) The atmospheric conditions. The mine had been closed for a few days and the air was perhaps noxious. (2) There was some new irritating property in the coal dust which, in combination with intense sweating, was capable of producing an irritant folliculitis. (3) The condition might be due to an infection in the mine with some fungus or organism.

The increasing number of industrial processes which utilize benzene in one form or another has brought in its wake a corresponding growth in the number of dermatologic industrial accidents. The summation of benzene accidents in the Paris Letter published by the *Journal of the American Medical Association*[31] is, therefore, timely. Benzene is the product of the distillation of either gasoline or coal tar. It is of some importance to know the source from which the benzene is derived as the products are not equally poisonous. In Germany, for example, "Benzene" means essence of petrol (gasoline). In France it means a distillation from crude coal tar which the Germans call benzol but in France benzol signifies a mixture of the hydrocarbons of coal. In the rubber and varnish trades there are more than twenty benzene solvents used, none of them alike. Of all these substances the pure benzene is the most toxic. It is not used in the pure state but mixed with toluene and xylene. Systemic poisoning may occur from the benzene products as well as dermatologic accidents.

As an example of the strange accidents to which the industrial worker is exposed may be cited the poisoning, which Henry[32] reports from celery. The cases were in workers who were preparing celery for canning. They developed a dermatitis confined to the upper limbs which itched somewhat and presented an erythematous vesicular or papular rash.

From time to time groups of signs and symptoms are separated from a larger and more general collection and are given new names. Haxthausen[33] has done this with a group which was once included in the general group of keratoderma. Gram once described a triad of symptoms encountered only in women at the climacteric, i.e., obesity, hypertension and chronic arthritis. To this symptom complex Haxthausen would now add a fourth, hyperkeratosis of the palms and soles. He has met this complex of obesity, hypertension, chronic arthritis, and keratoderma of palms and soles in ten women in the climacteric. The lesions in the skin were characterized by circumscribed keratoses, discrete, sharply defined, from a lentil to a pea in size, and devoid of symptoms. The lesions, through irritation, may become eczematized when the false diagnosis of a keratotic eczema may readily be made. In eight of his cases the systolic blood pressure ranged from 180 to 200. Treatment was disappointing. Haxthausen feels that the skin changes are due to endocrine disturbances associated with the menopause.

The arsenical keratoses are always interesting. McNeer[34] reports one case of arsenical keratosis and three of arsenical epidermoid carcinoma associated with keratoses. He states that it is the quinquivalent form of arsenic which produces keratoses and epitheliomas because of its predilection for ectodermal structures. The arsenical epitheliomata grow slowly, are of a low grade of malignancy and are but moderately radiosensitive. Metastasis may occur but it comes late in the disease. Treatment is a problem, he says, because it is often necessary to resort to several methods in the same case. Surgical excision is rarely practicable because of the multiplicity of lesions. The low voltage x-rays and mustard gas solution have given him the best results.

Every roentgenologist, and, for that matter, every well-informed physician, recognizes the dangers of x-ray therapy and for that reason is very insistent that none but capable and specially trained operators shall give it. In spite of that the x-ray has been put into commercial use notably in the removal of hair. The formation of carcinoma from x-ray burns is not uncommon in the literature. The formation of sarcoma is less usual. Yet it does occur as Kaplan's report proves again[35]. A woman, aged 28, was given two Roentgen treatments a week at the Tricho Institute for the removal of black hair on the face until she had had twenty treatments. Gradually the character of the skin changed. After five years a growth was noticed on the cheek. Two years later, seven years after the Tricho treatment, she was admitted to the New York Cancer Institute. It was noted on admission that there were present telangiectases and scarring on both sides of the face, the upper lip and the chin. On the right cheek was a strawberry red tumor mass measuring one inch by two inches and three-eighths inches high which was covered with a dirty, apparently in-

fected crust. At this time a diagnosis was made of epithelioma with secondary infection. The biopsy report was "fibroblastic granulation tissue". A radium pack was prescribed. The lesion failed to heal and one month later was removed by surgical excision. This time the pathologist reported "a spindle cell sarcoma".

Benatt and Hönighaus[36] have made a very interesting study of the action of saline baths containing carbon dioxide on the subpapillary venous plexus of the skin. They show that there exists a functional relationship between the subpapillary plexus in which there is stasis and which is present in mottling of the skin and other forms of acrocyanosis and the general circulation. It is known, the writers point out, that carbon dioxide saline baths produce a circumscribed hyperemia, increase the velocity of the circulation, counteract stasis and empty the dilated plexus. In a patient who had an extensive nevus flammeus, cool carbon dioxide baths effected a paling and even a temporary disappearance of the nevus. A temporary cure was obtained also in a number of patients with acrocyanosis. This improvement persisted in some instances as long as two years.

In the article just quoted we were dealing with a dilatation of the subpapillary veins. Dietel[37] writes of a spasm of the precapillary arterioles. During the cold season Dietel encountered a series of cases of peculiar whitish spots which at first sight simulated leucoderma. They were found more frequently in women than in men. Some patients showed a tendency to acrocyanosis. The white spots were distributed irregularly over the backs of the hands and over the forearms. They varied in size, but were never larger than a pea. If the arms were elevated for a time the spots disappeared. They could also be obliterated by rubbing, hot applications and the like, but reappeared soon. Cold intensified the spots and gave the skin a bluish-red color. Dietel believes that the cause lies in a vascular spasm in a precapillary arteriole which produces what is essentially an ischemia. The disease is related to cutis marmorata to a certain extent, but differs in that the retial pattern is lacking.

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CHRONIC ARTHRITIS*

BY FRANCIS COOLEY HALL, M.D.

If some of you who are listening in have been having joint pain for some time, or visible swelling about the joints, you probably call it arthritis, or you may call it by the older term of rheumatism. You may accept the condition with equanimity, or you may worry about it terribly, because of fear of being crippled. Your emotional reaction to this condition depends on your past experiences with the disease, and your emotional reaction determines what you will do about it. You may be doing nothing about it, you may be following a sensible régime of treatment under the guidance of a wise family physician, or if you have seen some relative or some Mrs. Smith become bedridden, you may rush from doctor to doctor, from one patent medicine to another, hoping for a quick cure. You may be right or wrong in the course you are pursuing. You may have joint pain but not have joint disease at all. You may have a type of arthritis which is annoying, which disfigures the fingers, which may give some limitation of activity in old age, but which will never cripple even though you have no medical care. Your neighbor, however, who is paying no attention to increasing pain and swelling of joints, who drives herself to keep going and kills the pain with drugs, may have a potentially serious and crippling type of disease. She is paying no attention to nature's warnings and may suffer serious joint damage before the disease can be controlled.

Thus, some of you need to be reassured as to your joint condition, while others need to be warned to take care of their joints and general health before damage is done that cannot be undone. In any case, joint pain or joint swelling is not a normal condition, nor are the other accompanying symptoms outside the joints normal, such as loss of appetite, and exhaustion. Mother Nature is trying to tell you something in giving you these symptoms, and urges you to find out what is wrong and what needs to be done about it. It is important not to wait too long before heeding the warnings.

Yet some of you are discouraged as to what can be done. Even some doctors are discouraged as well. It is true that damaged joints cannot be made well, though function can be restored by the orthopedic surgeon in many cases. There are certain patients with arthritis whose disease the medical profession cannot always control, even if treated early. However, quite as much can be done for patients with joint disease as for people with heart disease, diabetes, or other chronic diseases. The difficulty in the way of successful treatment of joint disease lies in the fact that it often is possible to ignore serious types of the disease until severe damage is done and the patient depleted because with the help of painkillers the patient may feel pretty well; yet various sets of statistics lead us to believe that in 70 per cent or more of people with true arthritis, the disease can be controlled, and the condition can be modified greatly in a still higher per cent. We are familiar with no sudden cures except where serious infection can be found and removed, but many people have attacks of arthritis which last only a few weeks and then cease regardless of the type of treatment. Arthritis not infrequently lasts for months and then ceases spontaneously. However, one cannot count on this, and careful medical study and care over a period of weeks and months is necessary in severe cases. Given proper opportunity and time, many doctors especially trained in the understanding and treatment of joint diseases feel optimistic about what can be done in controlling the disease for the average person suffering from joint pain or from arthritis, though in some instances, however, the patient may need medical supervision periodically for an indefinite period, as does a patient with heart disease, diabetes, or nephritis.

What kind of joint pain do you have? Does it represent arthritis or not? If it is arthritis, is it a serious type? I will summarize a few joint conditions in order to point out that joint pain or joint swelling may not represent a serious disturbance at all, but to point out *also* that it may represent a very serious disease beginning insidiously, with the power of increasing to the point of crippling.

There is a type of joint pain due to worry and emotional disturbance. There is another type due to overwork carried to the point of exhaustion. Overwork and worry often go together. Pain from either of these causes does not represent arthritis, and the pain disappears when worry disappears and the patient becomes rested. No joint swelling or changes in the joints by x-ray examination accompany this pain, though it is often called arthritis—quite incorrectly. It generally is not arthritis.

Joint and muscle pain are often associated with deficiency of certain of the glands of internal secretion, like the thyroid gland or the ovarian glands. The ovarian gland deficiency seems to be a factor in producing the symptoms of the change of life in women, and joint pain can represent one of these symptoms. These joint pains of glandular deficiency may not be serious, but can be most disturbing to the patient, and they may be more important than that, in being contributing factors in causing arthritis. The logical treatment of these glandular deficiencies is glandular medication. Such joint conditions may or may not represent true arthritis.

Joint pain and even arthritis can occur as the result of mechanical irritation of the joints due to blows, falls, normal use of joints through a lifetime to old age, and due to overuse or abnormal use of joints. Rest and protection of injured joints is most important if their integrity is to be restored, for continued irritation of an injured joint by using it before it is healed will lead to localized arthritis.

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Case Records

ANTE MORTEM AND POST MORTEM

AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

AT THE

MASSACHUSETTS GENERAL HOSPITAL

Published under the direction of

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CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.

CASE 20521

First admission. A twenty-nine year old Russian Jewish lawyer was brought into the Emergency Ward because of nausea and marked weakness.

During the week before admission the patient complained of a heavy lump in the midepigastrium which was present only at night. There was no heartburn or nausea. On the morning of admission he awoke feeling "mean". He had a large black bowel movement that morning following which he felt very weak and nauseated. That afternoon he vomited "quarts" of fresh red blood (perhaps a quart). Upon admission to the Emergency Ward he vomited a measured pint of fresh blood.

Physical examination showed a fairly well-developed and nourished man who looked acutely ill. The skin was warm, moist and pale. The heart sounds were of fairly good quality. No murmurs were heard. The blood pressure was 108/64. Further examination was not done because of marked hematemesis.

The temperature was 98°, the pulse 100. The respirations were 22.

Examination of the urine was negative. The blood showed a red cell count of 3,860,000, with a hemoglobin of 60 per cent. The white cell count was 25,000, 92 per cent polymorphonuclears. A stool examination showed a 4+ guaiac test. A Hinton test was negative.

During the first forty-eight hours he vomited a measured total of 64 ounces or a grand total of approximately 3,400 cubic centimeters of bloody material. On the third day he had his first transfusion. On the morning of the fifth day he vomited six ounces more of fresh blood and had another transfusion that day. The following evening he vomited about twenty ounces of blood. Immediately after this his pulse, which had been steady at 90, rose to 140, and his blood pressure dropped from 140 to 120. He was transfused on the seventh and eighth days and a final transfusion was done on the fourteenth day. There was no further hematemesis. He was given 3 cubic centimeters of liver extract intramuscularly on four different occasions together with 45 grains of Blaud's pills daily. His convalescence was uneventful.

X-rays of the gastro-intestinal tract showed only slightly thickened gastric rugae, and no cause for the hematemesis could be determined. A liver function test showed only 5 per cent retention. Examination of the blood during the third week showed a red cell count of 3,100,000, with a hemoglobin of 60 per cent, and a white cell count of 8,200. At this time the spleen was felt 7 centimeters below the umbilicus on quiet breathing. The liver could not be felt. It was learned at this time that he had had an enlarged spleen for seven or eight years.

He was discharged approximately one month after admission to return later for splenectomy.

Second admission. Five weeks later.

History of interval: Since his discharge he had been entirely well. He remained in bed only for a few days. There was no return of his former symptoms. He had eaten well and rested at home, preparing himself for a splenectomy. This was performed on the second day after admission.

On the sixth postoperative day the patient did not look well. He was listless and his cheeks were flushed. The pulse was 85 and the respirations were 18. The blood pressure was 138/85. Examination of the lungs showed increased whispered voice at the left apex anteriorly. The white blood cell count was 32,000. The next morning he had a sharp sticking pain localized in the right anterior chest unaccompanied by cough or sputum but with some cyanosis. The pain was constant even on the most shallow breathing and was relieved only by morphia. Examination of the anterior right chest revealed an area between the fourth and fifth ribs about the size of a quarter where the breathing was harsh and associated with an inspiratory friction sound. The next day he was slightly jaundiced and examination of the chest showed dullness to flatness, slightly decreased tactile fremitus, harsh bronchovesicular breathing and inspiratory râles at the right base posteriorly. The chest signs increased and were soon associated with coughing.

A portable chest film on the thirteenth postoperative day showed dullness in the left lung and the left side of the diaphragm obscured by the heart shadow. This dullness shaded out at the lower end of the scapula and the remainder of the left lung was clear. The right side of the diaphragm was high, sharp in outline, but the costophrenic angle was obliterated. There was a broad linear area of dullness overlying the lower angle of the right scapula which extended from the axillary line horizontally toward the heart. The heart appeared to be enlarged. Part of the changes at the bases of the lungs were interpreted as probably due to the high position of the diaphragm.

The red blood cell count ranged around 3,800,000, with a hemoglobin of 70 per cent. The white cell count averaged 22,000. The stools showed a negative guaiac, although the vomitus was positive.

On the twentieth day a chest tap showed hazy,

straw-colored fluid with a specific gravity of 1.014, 1,480 red blood cells and 720 white blood cells, 48 per cent of which were polymorphonuclears.

He gradually gained ground and was discharged approximately six weeks after admission.

Clinical Discussion

Dr. Beth Vincent: There is a great deal to be said about this case, but I shall confine my discussion to the surgical aspects and be as brief as possible.

In the absence of Dr. Minot, who had had this man under observation for seven or eight years, Dr. King asked me to see the patient in consultation at the time of the first massive hemorrhage. At that time it was evident that the patient was bleeding from esophageal varices or from the stomach and that nothing could be done except to try to keep him alive by transfusion, which to my surprise was successfully done, due to Dr. King's efforts. Six or seven transfusions were given. The patient was finally discharged from the hospital and we came to the conclusion that the probable diagnosis was either splenic anemia or thrombophlebitis of the splenic vein with splenomegaly. He was instructed to come back when in good condition at which time operation would be considered.

He was in excellent shape when he returned to the hospital, having had no bleeding since discharge. After considerable thought and discussion we decided to explore the abdomen and remove the spleen unless the liver was markedly cirrhotic or the vascular condition made splenectomy too dangerous.

The operative findings were very interesting. He had, as we had supposed, a large spleen. There were signs of old surface infarcts. The liver corresponded to that described in portal thrombophlebitis. It was smaller than normal but showed no cirrhosis to the naked eye. The most striking thing aside from the enlarged spleen was the collateral circulation that had been established between the spleen and the stomach by means of very numerous large veins in the gastrosplenic omentum and in the great omentum, which was adherent to the lower part of the spleen. The upper pole of the spleen was adherent to the diaphragm and these adhesions also carried large collateral veins. The engorgement of the veins of the stomach was the most marked I have ever seen, not only on the lesser and greater curvatures but in the wall of the stomach itself. On account of this mass of vessels it was not feasible to expose the splenic vein to determine its condition, but we came to the conclusion that this was a case of splenic thrombophlebitis or possibly of portal thrombophlebitis involving the splenic veins and other radicles of the portal system. The question then arose as to whether it was wiser to back out or to go ahead and try to render the patient less liable to another hemorrhage by interrupting the blood supply to the spleen and thus reduce the collateral circulation through the stomach and the esophageal varices. It was decided to go ahead and remove the spleen in spite of the very real danger of excessive bleeding or postoperative portal thrombosis. I tried to do it by first securing the vessels at the pedicle but the bleeding was so great that I finally resorted to the usual method of first mobilizing the spleen and then tying the pedicle. In spite of considerable bleeding the patient came through the operation fairly well with the aid of an immediate transfusion.

The convalescence confirmed the risk of this operation. He developed infarcts in both lungs, as shown by x-ray and physical signs. He had vomiting, diarrhea and abdominal pain which suggested mesenteric thrombosis but did not progress to the fatal point. He also had some superficial infection in the abdominal incision but returned to his home six weeks after the operation. The outcome of this case is uncertain. Whether we have diminished his liability to die of a massive hemorrhage or of developing a fatal mesenteric thrombosis I cannot tell.

Dr. Tracy B. Mallory: Dr. King, you saw this patient. Will you tell us more about him?

Dr. Richard B. King: We were very much interested in this fellow because his story goes so far back. He was first seen in 1926 in our office by Dr. Minot. The story then was that he had consulted Dr. Goodall in 1925 because he did not feel well. Nothing was found at that time, according to Dr. Goodall's records. He had no anemia; the blood was normal. No note was made of the spleen or liver at that time. He returned after a year to see Dr. Goodall, at which time the spleen was a good handbreadth below the costal margin. At that time his chief complaints were a feeling of heaviness and an occasional dull ache in the left upper quadrant. At that point he was referred to Dr. Minot for advice on the question of splenectomy. From the observations that Dr. Minot made, he could see no justification for doing splenectomy. The blood was normal. There was no anemia; and it is interesting that at no time during the eight years did he ever have anemia until this massive hemorrhage that he had in the hospital. The white cell count at no time was under eight thousand. There was an indefinite percentage of cells that were a little hard to classify, perhaps five or six per cent. The prevailing diagnosis in 1926 was a pseudo-leukemia and at that time we debated as to whether the spleen should come out, and whether he had Banti's disease. We saw him each year with the request from Dr. Goodall

that the spleen should be removed and each year we decided that it should not be removed. He had x-ray treatment by Dr. Morrison, followed by a note that the spleen had decreased in size, practically one-half of what it was before. That fitted in fairly well with the diagnosis of pseudoleukemia, whereas we could not explain why a spleen should decrease in size in Banti's disease with radiation. Our own observations are incomplete because there was a period of two years that we did not see him, but Dr. Goodall said there was a fifty per cent decrease in the size of the spleen with coincident loss of the unpleasant sensations that the patient had been having in the left upper quadrant. He went along uneventfully, as the history says, until this very dramatic episode took place. He was well, working hard, playing tennis and living a perfectly normal, happy existence until the vomiting of blood occurred.

The medical features of the postoperative course are the two pulmonary infarcts, one of fairly good size, the other small. He undoubtedly had a good deal of interference with circulation, as evidenced by the diarrhea, the vomiting, and the dull aching pains in the abdomen. He continued to run a fever as well as an elevated white count. About six weeks after operation he was considered well enough to be moved home, although he was still running a temperature from 99 to 101°. He was sitting up in a chair and walking around the halls. We felt that we had a bridge to cross and we might as well know if he was to have a fatal infarct. The family agreed. He went home two weeks ago.

During the first week after discharge he continued to run the same type of fever that he had had in the hospital. His appetite came back and he was sleeping well and had no pains. Four days ago the temperature became normal for twenty-four hours and he has had no fever since then. He walks three or four blocks a day and he feels better than he has ever felt. As Dr. Vincent has said, we can only guess at the outcome. I feel that in this instance the operation was justifiable on the basis that at any time he was apt to have a massive hemorrhage and not respond in such a lucky way as in this hospital entry. The last blood count showed 18,000 leukocytes and after splenectomy such a leukocytosis may last for a good many months. The infection in the wound has now subsided and is healing, with healthy granulation tissue; no skin graft is necessary.

Pathologic Diagnoses

Thrombosis of the splenic vein, organized.
Diffuse fibrosis of the spleen.
Infarcts of spleen, miliary, periarterial, hemorrhagic.
Splenomegaly.

Pathologic Discussion

Dr. Mallory: The spleen which was removed from this man was a very large one. I do not seem to have the weight recorded but the length was 28 centimeters and it must have weighed close to 1500 grams. On section there were one or two obvious anemic infarcts. The remainder of the spleen was somewhat pale, smooth, a little tough, and the markings were practically wiped out. We could not see any follicles or trabeculae.

The microscopic examination showed the diffuse fibrosis that is supposed to be characteristic of Banti's disease and also the acute progressive lesions that have been described recently by McMichael* in a larger series of cases of splenic anemia. They consist of hemorrhages about the small arteries, then organization of the hemorrhage with the deposit of considerable amounts of hemosiderin to make nodules of slightly brownish fibrous tissue that he speaks of as siderotic nodules. It also shows something I have never before noticed in Banti's disease but which McMichael mentions. In these foci are giant cells. They do not look like foreign body giant cells and are indistinguishable from megakaryocytes. Their significance in the pathogenesis of the disease is entirely beyond my power of explanation.

One thing that particularly interested me was the question of splenic thrombosis. Dr. Vincent had been unable to explore the region of the splenic vein at operation and as we looked at the pedicle grossly we could not find any recognizable splenic vein. We cut a number of sections, however, through the region of the pedicle and eventually demonstrated it, as I will show you.

This section is stained with a combination of the Van Gieson and the Verhoeff elastic tissue stains. Here is the edge of the capsule and here the tissues of the hilus with a branch of the splenic artery showing up very clearly. You will notice that all the tissue surrounding the artery contains innumerable small but dilated veins, at least fifty in that field, and the difficulties of the surgeon in attempting to control hemorrhage in a field like that are easily understood. The section also includes a bit of the tail of the pancreas, and the dilated veins are found all through it also. Here finally is a large vein, clearly outlined by the persistent elastic laminae even though it is four fifths blocked by old fibrous tissue, a completely organized and partially recanalized thrombus. At the upper margin there is a good sized canalizing vessel; along the lower margin several smaller ones. I think there can be no question that this man had years ago a thrombosis of the splenic vein. Had not multiple sections of the hilus region been made this case would never have been rec-

*McMichael, J.: The pathology of hepatolienal fibrosis. J. Path. and Bact. 39:481 (Sept.) 1934.

ognized as splenic thrombosis. This seems to me extremely important in regard to the relationship between splenic thrombosis and splenic anemia or Banti's disease. My own experience leads me to follow Warthin in assuming a causal relationship between the two conditions. It must be noted, however, that this man with the classical anatomic findings of Banti's disease had no anemia, except immediately after a severe hemorrhage, and no leukopenia at any time.

DR. KING: There is one other point. This man had a history of falling off a horse two or three months before he first consulted Dr. Goodall. We paid no attention to the story but it is interesting to speculate as to the possibility of injury to the splenic vein at that time.

DR. MALLORY: It seems to me that we have often heard that that is supposed to be found anatomically in Banti's disease, but from the clinical point of view there is only the large spleen and the hemorrhages to support it; no anemia and no leukemia. It can hardly be classed as a typical case of Banti's disease clinically; anatomically it is exactly as that is usually described.

A PHYSICIAN: Is there any evidence as to how long it takes collateral circulation to form in acute thrombosis of the splenic vein?

DR. MALLORY: We discussed but have never published one case here a year or two ago which gave some evidence on that. It was a woman who in 1907 had an acute infection associated with bloody diarrhea and the rapid development of a very large spleen. She was extremely ill for three or four months, then recovered, but the spleen remained large. Five years later she had three severe hematemeses in rapid succession, then lived twenty years with secondary anemia and a white cell count never over four thousand. At autopsy she showed a splenic vein nearly obliterated by thrombosis just before it entered the portal vein.

DR. ARTHUR W. ALLEN: I should like to ask Dr. Vincent one question, if I may. If this man's condition would have permitted it, would you have felt happier to ligate the vessels along the lesser curvature of the stomach as well, in order to protect him from further hemorrhage from the esophageal varices?

DR. VINCENT: I do not see how it would. I consider the collateral circulation in these cases as a relief from the obstruction in the splenic vein. But due to this need of collateral circulation we favor enlargement of the veins in the stomach or in the esophagus which may rupture and endanger the individual from massive hemorrhage. It seems more logical to me to diminish the flow of blood through the spleen by tying the splenic artery which in this instance I could do most easily by removing the spleen.

DR. ALLEN: You know what is in my mind, the work of Walters, who felt that if one were going to get relief from hemorrhages from esophageal varices not only the vasa brevia should be tied but also the vessels along the lesser curvature of the stomach; but in this case I can see how you could not superimpose further surgery on him.

DR. VINCENT: I look upon the collateral circulation as beneficent except that there is a tendency to rupture and death from hemorrhage.

CASE 20522

PRESENTATION OF CASE

A twenty-six year old white single school teacher entered complaining of a large mass in the left upper quadrant.

Approximately two years before entry the patient had a wart removed at a hospital and in the process of a routine physical examination it was discovered that she had a large spleen. This gave her no trouble at that time although she believed that she had been feeling tired and had fatigued easily for some time before that. Approximately nine months before entry she felt that the mass had become somewhat larger and produced pressure symptoms, especially when she lay down. She tired more easily and upon lying down there was some interference with complete inspiration. She had no other symptoms except for an occasional attack of indigestion.

Her father, mother, one brother and one sister were all living and well. The brother had arrested tuberculosis and her mother had had tuberculosis in youth.

The patient had lived in New England all her life. As a child she had measles, mumps, chicken pox and pertussis. There was no history of pneumonia or pleurisy. She had had edema when four years of age but had no recurrence since. Nine years before entry she had a tonsillectomy and an adenoidectomy because of repeated sore throats. She believed, however, that her sore throats had increased since the operation. She usually had two or three sore throats during the winter, each lasting about three days and not associated with chills. Eleven years before entry she was told that she had a valvular defect of her heart but she never had any symptoms of decompensation. Her menses began at thirteen years of age and occurred every twenty-eight days, lasting six days and requiring about a dozen pads in all. She had always had severe cramps but they were never severe enough to prevent her from performing her usual activities.

Physical examination showed a healthy appearing girl in no apparent distress. There was a questionable slight icteric tint to her sclerae. No glands were felt anywhere. Abdominal ex-

amination showed marked enlargement of the spleen to the level of the umbilicus, with a palpable notch. It was not tender. The liver edge was felt one fingerbreadth down upon inspiration. There were a few areas of ecchymosis on the legs and arms. The blood pressure was 120/70.

The temperature was 99.6°, the pulse 85. The respirations were 22.

Examination of the urine showed a slight trace of albumin and a specific gravity of 1.028. The blood showed a red cell count of 4,850,000, with a hemoglobin of 80 per cent. The white cell count was 9,200, 87 per cent polymorphonuclears. The icteric index was 5. The liver function test showed less than five per cent retention of the dye. A Hinton test was negative.

An intravenous pyelogram showed that the left kidney outline was longer and lower than the right. The intravenous dye appeared promptly in both kidney pelves. The right kidney pelvis was larger than the left. There was suggestive evidence of infection in the right kidney. The splenic shadow extended nearly to the iliac crest.

On the seventh day a splenectomy was performed and a biopsy of the liver taken. A blood smear postoperatively showed one striking change over the preoperative picture. The platelets now appeared in large clumps many of which were larger than the total cytoplasm of megakaryocytes. On the third postoperative day she developed abdominal distention and marked tenderness. Her temperature rose to 104°. Toward the end of that day her blood pressure fell and her pulse rose. That evening no blood pressure was obtainable. She was put in the shock position and was revived somewhat after intravenous coramin. She gradually became more cyanotic and died about five hours after an apparent circulatory collapse.

Clinical Discussion

Dr. Arthur W. Allen: This patient appeared at my office one day and when I asked her what I could do for her, never having seen her before, she said "I would like to have you take out my spleen." It is the only time that such a request had been made of me.

This patient was seen some six weeks prior to this admission by Dr. Bock, who will tell you of the findings at that time. After she was sent in here Dr. Vincent saw her with me, and also Dr. Francis Hunter. It was finally decided that splenectomy was justifiable and it was done.

The operation itself was moderately difficult, as these spleens are apt to be. The vasa brevia were enormously dilated. There were a great many anastomosing enlarged veins at the lower pole of the spleen. There were a few of these large collateral veins also in the diaphragmatic bed but not so many as in some cases. I was not able to identify the splenic vein at all. If there was a splenic vein it was very much divided or contracted and as a separate entity I could not identify it. The operation did not seem to bother the patient too much. She was in good condition at the end of it and all the hemorrhage was easily controlled. However, she did not look very well the day after operation. She was a little more alert, a little more restless than the average abdominal case should appear on the day after operation. The second day after operation she also looked ill, and her pulse began to rise. Her abdomen became somewhat distended, and in spite of the fact that audible peristalsis could be heard with the stethoscope we thought she might be getting peritonitis, which may occur after splenectomy, possibly due to interference with the blood supply to a hollow viscus, allowing perforation to occur. She had a rapid exodus. The terminal events were circulatory collapse and cyanosis.

Dr. Arlie V. Bock: I saw this girl approximately one month before she entered the hospital, when she appeared to be in quite good condition. There are just one or two things about the physical examination upon which I should like to comment. The note says that there was a questionable slightly icteric tint to the sclerae. It must have been very questionable. When I saw her I thought the sclerae were quite white, pale, suggesting anemia. There was no evidence of valvular lesion in the heart. The abdomen showed fullness of the left side. The left half was dull to percussion, the right half showing normal tympany. The edge of the spleen could be felt well across the midline in Traube's space and extended below the level of the navel. I could not make out a notch. The flank was full, and it seemed certain that we were dealing with a spleen and not a kidney on the basis of that finding. A big kidney usually fills the whole flank. When I percussed it the dullness went out and well up in the left side in the region of the fifth space and made an "S" line around to the back, an outline one might expect from a big spleen. In the left lateral position one could more readily obtain the outline of the edge of the mass but even then I was unable to make out a notch.

The thing that made me feel that splenectomy would be very difficult in this case was the fact that this huge mass if it was the spleen was firmly fixed and suggested a great deal of perisplenitis with adhesions of some magnitude to the diaphragm. The whole picture suggested a very difficult operative procedure.

The blood surprised me in this respect; she had a red cell count of 5,600,000 and a white cell count of 8,000. The smear showed 78 per cent polymorphonuclears, 15 per cent lympho-

cytes, 1 eosinophile, 1 mast cell and 5 large mononuclears; all of the cells were normal white cells. The platelets were somewhat reduced in number and otherwise seemed normal. The red cells showed no achromia. The bleeding time was normal, coagulation time eight minutes, and the clot retraction was normal. The next day the serum was of normal color. At that time I wrote to Dr. Allen and said that I did not think this was a Banti's disease, reasoning on the same basis that Dr. King and Dr. Minot had in the previous case, because I was not familiar with a Banti's syndrome with a high red cell count and a normal white cell count such as this girl showed. On looking up all the cases of Banti's disease with splenectomy for a period of ten years in the Massachusetts General Hospital I have found fifteen cases, eight in women and seven in men. Two or three of them had high red cell counts and normal white cell counts. Of these fifteen, seven had no history of hemorrhage, eight had had hemorrhages; three cases were under thirteen years of age. I was also interested in the comments of the surgeons at the time of operation. There is only one case in which the surgeon failed to mention the condition of the liver. In three others there was a question of a possible mild cirrhosis and in the others no suggestion of cirrhosis at all. I had always been taught to believe that a Banti's syndrome led eventually to cirrhosis but I believe that is a mistaken idea and the conception that we now have, that Banti's disease or so-called splenic anemia is the result of thrombophlebitis of the splenic vein, is more probable. The circulatory obstruction is the main factor in producing the enormous enlargement of the spleen in these cases with the consequent development of a huge collateral circulation. On this basis it seems to me that we have a very logical justification for doing splenectomy, so preventing an amount of blood getting into the spleen bed which is unable to go out satisfactorily through a thrombosed vessel. Collateral circulation develops with eventual rupture from the varices. If one removes the spleen and the surgeon is able to do this successfully one has relieved the portal system from an enormous load. I think the history in a great many of our cases has suggested remarkable relief from subsequent hemorrhages. That is not true of all of our cases in which the spleen has been removed. Hemorrhages may occur from more thrombosis in the portal system. I think now that this was a case of Banti's disease.

Dr. Tracy B. Mallory: Dr. Hunter, you saw this case. Have you anything to add?

Dr. Francis T. Hunter: I think Dr. Bock has said all there is to be said about it. The only thing I might bring up is that this case illustrates the danger of doing too careful a physical examination.

Dr. Bock: There is one other point I should like to mention. This was a twenty-six year old girl with a mass in the upper quadrant and I practically insisted to Dr. Allen that we do a pyelogram to rule out kidney. That may seem to be taking extra precaution but I have seen at least one case in this hospital where the entire medical service, including Dr. Vincent, made a diagnosis of an enormously enlarged spleen. A diagnosis of Banti's disease was made and operation advised and attempted by Dr. Vincent. What we found was an enormous primary carcinoma of the left kidney. In Dr. King's case I think they were justified in going ahead with splenectomy, in view of the history of the hemorrhage, without doing a pyelogram; but in this case there had been no history of hemorrhage and we had a possibility of other pathology, even though the physical examination strongly suggested spleen. I should not have been content to have Dr. Allen explore her without knowing about the kidney.

Clinical Diagnoses

Splenomegaly.
General peritonitis.

Anatomic Diagnoses

Old thrombosis of the splenic vein.
Splenomegaly.
Diffuse fibrosis and periarterial hemorrhages of the spleen.
Acute thrombophlebitis, mesenteric, portal and splenic veins.
Acute central necrosis of the liver.

Pathologic Discussion

Dr. Mallory: The autopsy on this girl showed when we first opened the abdominal cavity that the entire small intestine from the jejunum down to the ileocecal valve was brilliant red, and on cutting into the mesentery it was evident that every branch of the superior mesenteric vein was thrombosed. We could follow the thrombi upward into the portal vein, laterally through the remaining remnant of the splenic vein, then on into the various radicles of the portal vein within the liver. There was no part of the portal system that was not completely obstructed. We looked with a great deal of care at the time of autopsy for any evidence of old thrombosis of the splenic vein but we were unable to find any. The fresh thrombosis extended directly up to Dr. Allen's ligatures. The spleen itself unfortunately was not examined in the way the spleen in the preceding case was, but since neither we nor Dr. Allen could find a splenic vein I think we can fairly assume it must have been obliterated. I believe if we had made multiple sections through the hilus region after we were unable to identify the splenic vein in gross,

we would have picked up histologic traces of it as we did in the first case.

The literature on Banti's disease and splenic anemia is extraordinarily confused. It is very difficult to make out with what frequency thrombosis of the splenic vein has been found. Certain people, notably Warthin*, have found it in every case personally examined. It is evident that a thrombosis if it is an old one may have resulted in so much obliteration and distortion of the architecture that it might be far from easy to discover the thrombus. It may occur at any point in the vein but a most important place to look for it is the hilus of the resected spleen. The fact that it is not found at autopsy after splenectomy has been done does not at all rule out its presence. This acute massive thrombosis of the portal system is of course the standard complication of splenectomy. If they do not develop portal thrombosis they stand an excellent chance of recovering from operation but a very considerable proportion of them will die of this condition and so far as we know there is no way of preventing it.

DR. ALLEN: I should like to know if there is an optimum time of operation on such a patient with thrombophlebitis of the splenic vein. Should we wait on these cases until they have had a subfatal hemorrhage before doing anything about it, or should we urge them to have splenectomy at the earliest possible moment the diagnosis can be made?

DR. MALLORY: In some work done at Mt. Sinai it was claimed that they can be divided into two groups. In some of them the platelets are elevated, while in others they are diminished. If you operate on a case with elevated platelets you will almost certainly get thrombosis; in the other cases it is much less likely.

DR. HAMPTON: I should like to ask Dr. Hunter if he thinks x-ray would affect the platelets before operation, that is x-ray treatment of the spleen?

DR. HUNTER: I do not know.

DR. MALLORY: The liver showed no cirrhosis. There was a very early acute central necrosis which I am sure was subsequent to the portal thrombosis.

DR. VINCENT: Do you think this was a splenic anemia, such as Osler described, due to intoxication of unknown origin, or the type due to thrombophlebitis of the portal vein or its radicles?

DR. MALLORY: I think there is very little doubt that it was due to thrombosis of the portal veins.

*Warthin, A. S.: The relation of thrombophlebitis of the portal and splenic veins to splenic anaemia and Banti's disease. Internat. Clin. 4 (Series 20): 189, 1910.

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PSYCHOANALYSIS

PSYCHOANALYSIS is approaching its fortieth birthday, Freud having first presented his preliminary investigations at a meeting of the Vienna Medical Society in 1896. For ten years thereafter the founder worked alone, practically ostracized by his professional colleagues. Gradually students came to him. By 1907 collaboration of his investigations was undertaken; in 1909, the first psychoanalytic journal was published; the next year, an international society was formed. Some idea of the extent of psychoanalysis to-day is gained by noticing that eight journals are now issued, three in English, and that societies for study of this particular field are established in many countries. Books on psychoanalysis — informative, historical and critical—are published without number. Those, recently issued in English, of most importance are "Facts and Theories of Psychoanalysis" by Ives Hendrick[1], "Psychoanalysis and Medicine" by Karin Stephen[2] and "New Introductory Lectures on Psycho-Analysis" by Sigmund Freud[3].

The serious student, in spite of the vast amount of literature available, still has considerable doubt about "psycho-analysis". Is it a science, a psychological method of investigation of the unconscious functions of the mind; a theory, often twisted by the glamour of Freudian worshipers; an experimental problem or a therapeutic measure; a cult, distorted by a band of unscientific followers?

Dr. Hendrick, a sound disciple of Freud, answers some of our questions in his clear, practical book. Psychoanalysis is, according to Hendrick, "a science of the unconscious functions of the mind and personality, developed by Sigmund Freud and his students." It is not synonymous with any type of psychotherapy or sexual psychology in general. The theory is based upon what the psychoanalysts consider as a fact: namely, "that adults remember much of their lives after five years, and a very few experiences of earlier years" and, therefore, "the human race forgets the events of infancy because infancy is a period when sexuality and rivalry are taboo, and yet is unconsciously influenced by them in adult life." It is here that all cannot follow. Hendrick gives an excellent summary and marshals his arguments in good form, but can we be sure of his basic postulates? To the ordinary physician it would seem that many more published empirical observations are called for, before the basic theory is completely acceptable. Advancement calls for research with sound men subsidized to carry it out. Too many psychoanalysts are forced to "practice" to-day, using methods still in the laboratory stage of investigation. To some of true scientific bent this is distasteful; to the peripherally placed followers of the theory, it has proved to be a profitable business. Psychoanalysis has suffered greatly by commercialism.

We must face the problem broadly and squarely. A growing subject, it should be controlled by the medical profession as long as it has therapeutic possibilities. If it has none, as many think, it belongs in the field of psychology. Medically, we need more data in regard to the results of treatment. Criteria of cure are difficult to establish, particularly so in view of the fact that there is considerable difference of opinion as to the road on which we travel and the goal which we are attempting to reach. The reports are growing, but medicine must wait, perhaps for many years, before either the theory receives world-wide acceptance or the therapeutic method is accepted.

REFERENCES

1 Hendrick, Ives: Facts and Theories of Psychoanalysis. New York, 1934.
2 Stephen, Karin: Psychoanalysis and Medicine. New York, 1933.
3 Freud, Sigmund: New Introductory Lectures on Psycho-Analysis. New York, 1933.

THE ENTERTAINMENT OF LADIES AT THE ANNUAL MEETING

At the Annual Meeting of the Massachusetts Medical Society to be held June 3rd, 4th and 5th, it is hoped that the wives and daughters of Fellows will be interested in the hospitality which is being arranged by a committee of women. On each of these days there will be an event at which ladies will be guests of the Society. A tea on Monday at the home of Mrs. Randall Clifford, a tea on Tuesday at that unique spot in America — The Isabella Stewart Gardner Museum, Fenway Court; a trip to Concord on Wednesday, including lunch at the Country Club, and an opportunity to see one of the most charming gardens in that vicinity.

Arrangements are being made so that ladies who are interested may visit the Boston hospitals.

If the ladies of your family respond to these invitations it will serve to arouse a great sense of friendliness and will increase that spirit of camaraderie which was such a pleasant feature of the Worcester program.

Notice of these arrangements is being given well in advance in order that plans for early June may be made with these events in mind.

The Women's Committee would like to stress the fact that the hospitality is offered not only to those coming from a distance but to the wives and daughters of doctors in and about Boston. In addition, ladies are cordially invited to attend the Shattuck Lecture Monday evening, June 3rd, when Dr. William E. Gallie of the University of Toronto will speak.

Will you kindly show this to your family NOW?

COMMITTEE

Mrs. William H. Robey, Chairman,
Mrs. Roger I. Lee, Vice-Chairman,
Mrs. Herrman L. Blumgart,
Mrs. Frederick L. Good,
Mrs. Elliott P. Joslin,
Mrs. George R. Minot,
Mrs. George P. Reynolds,
Mrs. William M. Shedden.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

Sever, James Warren. M.D. Harvard University Medical School 1901. F.A.C.S. Associate Surgeon, Boston City Hospital and Boston Children's Hospital. Assistant Professor of Orthopedic Surgery, Harvard Medical School. His subject is "Slipping Epiphysis of the Head of the Femur." Page 1179. Address: 321 Dartmouth Street, Boston, Massachusetts.

Lyon, James Alexander. M.D. Maryland Medical College 1906. Graduate Study at Harvard Medical School; National Heart Hospital; London, and University of Vienna. Professor of Clinical Cardiology, Georgetown University School of Medicine, Washington. D. C. Attending Cardiologist Emergency, Columbia, and the Children's Hospitals, Washington. His subject is "Rheumatic Heart Disease in Early Childhood." Page 1185. Address: 1801 Eye Street, N. W., Washington, D. C.

Walker, William G. M.D. State University of Iowa Medical College 1919. Formerly, Junior Associate in Medicine, Peter Bent Brigham Hospital. Physician to The Brockton Hospital since January, 1924. His subject is: "Coarctation of the Aorta in Father and Son." Page 1192. Address: 12 Cottage Street, Brockton, Massachusetts.

Fishberg, Arthur M. A.B., M.D. Columbia University College of Physicians and Surgeons 1921. Associate in Medicine, Mount Sinai Hospital. Associate Physician, Beth Israel Hospital, New York City. His subject is "Clinical Remarks on Hypertension and the Kidney." Page 1195. Address: 1136 Fifth Avenue, New York City.

Aschner, Paul W. A.B., M.D. Columbia University 1912. F.A.C.S. Attending Urologist, Hospital for Joint Diseases, New York City. Member, American Urological Association. Fellow, New York Academy of Medicine. His subject is "Indications for and Principles of Surgical Therapy in Disorders of the Kidney and Ureter." Page 1196. Address: 12 East 88th Street, New York City.

Towle, Harvey P. A.B., M.D. Harvard University Medical School 1892. Consultant in Dermatology, Massachusetts General Hospital. Attested by American Board of Dermatology and Syphilology. Address: 453 Marlboro Street, Boston, Massachusetts. Associated with him is

Grund, J. L. M.D. Harvard University Medical School 1921. Assistant Dermatologist, Department of Dermatology and Syphilology, Massachusetts Memorial Hospitals. Instructor in Dermatology and Syphilology, Boston University School of Medicine. Address: 483 Beacon Street, Boston, Massachusetts. Their subject is "Progress in Dermatology 1934." Page 1200.

MISCELLANY

THE REAPPOINTMENT OF DR. MAHONEY

Mayor Mansfield has reappointed Dr. Francis X. Mahoney as Commissioner of Health for the City of Boston for the term ending April 1, 1938.

Dr. Mahoney has occupied this position since 1922.

IS THERE UNUSUAL PREVALENCE OF RESPIRATORY DISORDERS?

Physicians in Massachusetts are reporting cases of an acute febrile disorder with diarrhea in some instances and irritation of the respiratory tract in others. Most people speak of the malady as a cold. That diagnosis is a confession of ignorance; for the cause of the so-called cold has never been discovered.

In New Jersey a very prevalent acute febrile disorder is referred to in the daily papers as "grip" or "influenza" which has led to the closing of a number of public schools, and the State Department of Health of New Jersey has issued rules of hygiene to check the spread of the contagion. The Health and Education Departments are recorded as having announced that "Grip has suddenly become unusually prevalent, particularly in the southern section of New Jersey." Parents and guardians have been urged to have the children who are ill provided with medical care. Thus far a few cases of pneumonia have been reported as complicating the illness.

Although hundreds of children are ill, the extent of the epidemic is unknown, because under the designation of grip there is no requirement for reporting the cases.

Long Island also reports hundreds of cases which have led to the closing of three schools.

Dr. Rice, Commissioner of Health of New York City, is reported to have said that although the seasonal increase of respiratory disease is present, there is no epidemic of grip or influenza.

CHRONIC ARTHRITIS

(Continued from Page 1208)

If taken care of, these injured joints may become quite normal. Joint pain is especially common in people who stand and walk incorrectly, who use their joints "out of line". Such people develop pain in the joints and in the muscles and tendons about the joints at the *end* of the day, but this pain disappears after a night's rest. The back, neck, knees, and feet are especially prone to this type of pain. As the years pass, this misuse of the joints may cause joint injury, or arthritis. Early, this joint pain is *not* arthritis, but joint strain. The treatment is correction of swayback, flat feet, and bad posture as a whole. Complete relief of pain generally follows. Overweight people with painful knees are in this group. They need to be properly balanced as to knees and feet and to lose weight.

People have joint pain associated with colds, influenza, or any infection anywhere in the body. There may be joint pain associated with almost any disease, bad diets, or poor gastro-intestinal function. In these conditions, toxemia from bacteria, allergic reactions to protein in bacteria and in food, some malnutrition effect from badly balanced or deficient diets are the causes of the joint pain. Frequently these joint pains do not represent arthritis at all, but these same causes may be responsible for or condition definite joint disease, especially if neglected. Thus, joint pain may not represent arthritis at all, but may signify disease in the body, or bad function of the body in some of its parts. But again, joint pain may represent early arthritis. It is not easy to determine whether these conditions will lead to severe arthritis or be of minor importance. If the disease with which the joint disturbance is associated is properly treated—not only by the doctor, but by a coöperating patient, both the disease and the joint condition will subside in most instances. If swelling as well as pain is ignored, and the joint further inflamed by use, and the body further depleted by carrying on to the point of exhaustion, a case of chronic, progressive arthritis is in the making.

The more severe types of joint pain, associated with swelling, inflammation, and marked disability are rheumatic fever, gout, and infectious arthritis. *Rheumatic fever* occurs especially in children and young adults, is accompanied by fever, hot, red, swollen, and very tender joints. These eventually subside and become quite normal again. The danger from rheumatic fever is damage to the heart. *Gouty arthritis* is far more common than is generally appreciated, and it is not necessarily associated with high living. It comes suddenly, especially in the foot or knee, and though it is actually painful, it subsides in a few days or weeks, only to reappear weeks or months later, unless controlled by a low purine diet. *Infectious arthritis* may be severe and crippling, or it may be mild and of short duration. It may be caused by almost any infection, or follow infections like tonsillitis, scarlet fever, and pneumonia. Its cause may be carefully cared for like any sick person until well. This is one of the types to worry about, for, unless properly cared for, a condition of chronic progressive arthritis may result.

There is a type of arthritis called Rheumatoid or Atrophic Arthritis whose cause is not always clear. It is generally considered infectious in origin and may develop slowly in people under forty-five who are run down and depleted. It may develop gradually with pain on waking in the morning, but improve as the day wears on. It is associated with soft swelling of the middle joints of the fingers, or the wrists, or elbows, knees, or spine. It occurs especially in people who drive themselves to the point of exhaustion. Infection can generally *not* be proved. These people are gravely ill and need prolonged study and the type of care which builds up their general health in every particular, rest, diet, special medication, exercises, and attention to the body as a whole, as well as to the joints.

The last type of arthritis of which I wish to speak is the hypertrophic type. This occurs in most very old people and in many over fifty. It may be painless and only noticeable because of hard enlarge-

ments of the fingers, or there may be stiffness, or even much limitation of motion in the joints. This type represents the ageing, wearing-out process. It is usually not serious, but may cripple to a degree, especially if present in the hips or knees. Mechanical irritation of the joints from use, poor joint nutrition associated with ageing, glandular deficiencies, bad posture, and incorrect diets are some of the conditions found which are thought to be conditioning factors. Some of these joint conditions can be markedly improved, while other conditions can be controlled only as one controls a patient with heart disease — by keeping him within prescribed limits of activity.

Now whether you have joint pain or swelling, the most important thing you need is a careful medical study of yourself as an individual, and a precise diagnosis of the nature of your joint trouble with a rational plan of treatment. And remember your "neuritis" may represent arthritis, your headache may be due to trouble in the neck, your "pleurisy" may be due to arthritis of the upper spine, and the pain in the legs may be due to damage taking place in the hip joints. Whatever you do, remember that (continued) joint pain, muscle pain, and swelling represent real sickness, and you should give the body a chance to heal. Get more sleep, a better diet, with more milk, eggs, green vegetables, and fruit, and if you are overweight, less starches and sugar. Don't give up meat unless you have gout. Take daily exercises, without using the sore joints to the point of making them worse. Above all, stop taking pain-killing drugs, for these make it possible for you to use and thus to irritate inflamed joints, and don't try to continue to use the joints despite pain until nature forces you to stop; you can't beat nature by sheer will power.

THIRTY EYE SPECIALISTS INSPECT AMERICAN OPTICAL COMPANY'S PLANT

Thirty oculists and ophthalmologists from Boston, Worcester, and Providence, R. I., visited the American Optical Company, November 22, as guests of company executives and inspected the developments in refracting instruments and equipment.

They were welcomed at the plant by Channing M. Wells, president of the company.

Dr. Julius Neumueller, associated with the optical concern and now doing research work at Dartmouth College, spoke on "The Latest Developments in Anseikonia and Iseikonic Lenses."

Dr. Albert Bielschowsky, a German ophthalmologist who is studying at Dartmouth, was the guest speaker.

Ira Mosher, general manager of the optical company, also spoke.

The visitors were entertained at luncheon with the company officials and then were shown through the lens plant.

CORRESPONDENCE

OFFICIAL ACTIONS OF THE BOARD OF REGISTRATION IN MEDICINE

Board of Registration in Medicine
State House, Boston

December 22, 1934.

Editor, *New England Journal of Medicine,*

This is to inform you that at a meeting of the Board of Registration in Medicine, held December 20, 1934, the Board voted to revoke the license of Dr. R. Reyes Garcia, of 353 Dwight Street, Springfield, Massachusetts, for deceit in the care of a patient. At the same meeting the license of Dr. S. Margaret Brown of 22 Blagden Street, Boston, was revoked for deceit in the care of a patient.

Very truly yours,

STEPHEN RUSHMORE, M.D., *Secretary.*

MEDICAL ECONOMICS

Editor, *New England Journal of Medicine,*

"Medical Economics"! The words seem to saturate the very air we breathe, producing a condition bordering on anoxemia, as is evidenced by "the blue in the face", when we talk about the various ills and panaceas which confront us at present. We seem to be awakening to a none too pleasant sensation, that somebody is trying to injure us in these troubled times. We are awakening, but we are not yet fully conscious as to what it is all about, and we stand aghast trying to grasp the meaning of existing conditions. Whether one meets a colleague, attends a medical meeting, or reads a journal, the question is always there. What is happening to our profession? Whither are we drifting?

The lay press is bombarding us on all sides with its silly accusations, arising chiefly from petty jealousies, fond theories and gross misinformation about the subject of medical practice.

It is true that here and there a voice is heard against this unjust onslaught upon our profession. Now and then the more wakeful report abuses heaped upon us. But, besides reciting and listening to grievances, and in the end feeling the worse for it, little is done about it. It often reminds one of a postmortem clinic, where data are presented, diagnoses made, each one given a chance to take part in the discussion, but the patient remains dead, nevertheless.

What then is to be done about our precarious situation? Is it perhaps too late, as so many are inclined to believe, to enter action that will help to overcome the impending crisis? What can be done to improve our dwindling practices, to raise our shrinking incomes, to show up the calumnious attacks of the press; and how can we use the radio to the advantage of the public and ourselves? What can be done to prevail upon hospital authorities to stop the abuse of free clinics, crowding out the worthy poor, cutting into our incomes, and adding

to the burdens of the taxpayer? How can we stop the activities of boards of health, and encroachment upon our private practices? How can we bring social service and other agencies back to the place for which they were originally intended? And above all, how can we show a misinformed public, that in order to satisfy the whims and fancies of many agencies and philanthropists medicine is being made the goat in a game called "socialized medicine", a game that may yet ruin us as a profession, and destroy that "doctor and patient" relationship without which the practice of medicine would be a farce?

The situation is disconcerting but there must be a solution, provided we go about it in a logical, businesslike way. And since the underlying causes of nearly all our troubles are economic, the way to solve them must lie along economic lines, and for that we must follow the practice of the big corporation where economic questions are never disregarded, but handled as the basis of the lifeblood of the business. For instance, a corporation finds itself in some unusual critical condition; the heads of the firm do not attempt to solve it by meetings of the entire stockholding membership. That is, they may take the shareholders into their confidence, but the actual working out of a program to meet the particular situation is left to such experts in the business as are specially qualified to deal with the problems.

From this, it is apparent that we must assume similar methods if we are ever to arrive at a solution of our own ills. These methods, however, must be so modified as to suit our already existing organization.

This is how it can be done: Let each individual medical unit select one or two of its members, who are well known for their willingness to do something for their colleagues. They may be appointed by the chair, but, preferably, they should be elected by the members at large for their ability to deal with economic questions. These members should be known as delegates to "a medical economic club". The members so elected from the various branches of a district should constitute "a district medical economic club" and they in turn should form a "state medical economic club". The district delegates so elected and so named should meet at frequent intervals, to receive claims or suggestions from individual members of their respective societies. These suggestions and grievances should be taken up at the district medical club meetings and acted upon there and then. Weightier problems could be presented at "state economic club meetings", the latter to be arranged from time to time. In this way, the views of the profession at large would find coherent expression; various reports of abuses as reported by individual physicians could be tabulated; measures, such as taking care of the indigent poor, or actions against false propaganda for socialized medicine would thereby find reasons for proper action. And in a general way, we would be prepared for any economic emergency that might arise. Only a medical club so elected would be a truly representative body of the members of our medical societies, and only such a club would be in a position to reflect our views and formulate our desires.

The individual members would feel that they are truly represented on an economic field, and problems would be more amenable to consideration and solution than is the case at present. Also actions would become more uniform and more obligatory upon the individual members. At the same time, a section in our state medical journals could be devoted to the workings of the "medical economic clubs" keeping the membership informed of their decisions.

To summarize: In order to improve our present-day practice, in order to fight off the various evils confronting our profession, we must be organized on economic grounds, and we must have a body of men that will truly represent the rank and file of our organizations. "An economic medical club" so elected will be truly representative, and will ultimately be so constituted as to bring about real and constructive results.

Very truly yours,

R. GURALNICK.

256 Bennington Street, East Boston, Mass.

FOTHERGILL'S OBSERVATIONS ON ANGINA PECTORIS

Editor, *New England Journal of Medicine*,

The following quotation is from "Dr. John Fothergill and His Friends," by Dr. R. Hingston Fox. (Macmillan Company, London, 1919.)

It is of interest that the particular volume from which I quote was lent to me by Mrs. E. L. White of Montreal, a direct descendant of Dr. Fothergill. Although his works are well known to many internists, his classic description of angina will, I think, be of interest to practitioners at the present time, when the study of cardiac disease, especially angina, is so much to the fore.

Hingston recalls that this cardiac syndrome had been defined and named by Heberden, though isolated cases could be found in the records of Morgagni and others.

Fothergill, we are told, described two cases out of many observed by him, where he had been able to procure a *postmortem* examination.

"The clinical history is related with fullness by his graphic pen, including the special characteristics of the pain, the effect of sudden agitations, whether from passion or exercise, the influence of a full meal, and of states of the weather, and the frequent association with a gouty habit of body. Of the autopsies on his two patients, the first was inconclusive, the second was performed by John Hunter—it might have been a forecast of his own tragical end—and recorded besides pleural effusion, much evidence of 'ossification' in the structures of the heart. The substance of the ventricles was pale, ligamentous and in part whitish and hard; the mitral valve was stiffened and contained many similar patches; there were ossifications in the wall of the aorta; and 'the two coronary arteries, from

their origin to many of their ramifications upon the heart, were become one piece of bone.'"

Hingston remarks that "This seems to be the earliest dated record of lesions of the coronary vessels in a case of angina pectoris. Jenner and Parry made further observations, and coronary atheroma and its results were in course of time recognized as the characteristic lesions of one type of angina, whilst Heberden's original theory of spasm was developed by Trousseau and others."

Fothergill's scheme of management of angina, Hingston further says, "would still form a good basis of treatment, to which the further and more potent means which modern knowledge has supplied could be added." The papers, he states, "seemed to have been read in 1774 and 1775."

Very truly yours,

WM. PEARCE COUES, M.D.

12 Monmouth Court, Brookline, Mass.

RECENT DEATHS

DEATH OF DR. F. RUSSELL DAME

Dr. Fred Russell Dame, of 1032 Main Street, Athol, was instantly killed Friday, December 21, 1934, when an automobile, in which he and his son were riding, was hit by a locomotive on a crossing in Athol. Dr. Dame had driven to Amherst to bring home a son, Frederick, who attended Amherst College. The accident occurred a short distance from his home.

The son, who drove the car, was only slightly injured, whereas the doctor's death, from a fractured neck, was instantaneous. The driver had stopped at the crossing to allow an express train to pass and then drove in the path of another train going in the opposite direction.

Dr. Dame was born in Farmington, N. H., in 1872. After graduating from the Baltimore Medical College he practised in Medford and Braintree for many years, moving to Athol ten years ago, establishing a large practice. He was Past Master of Athol Lodge, A. F. and A. M., Royal Arch Chapter.

He was a member of the Council of the Massachusetts Medical Society and most active in the deliberations of the Worcester North District Medical Society. He rarely missed a meeting, being a strong supporter of the ideals of the state society, and maintained a high degree of professional ethics. He will be sadly missed at the quarterly gatherings.

Surviving him are his widow, Mrs. Helen F. Dame; two sons, Frederick and Elmer, and a sister, Mrs. Emma Libby of Amesbury.

The manner of his death, the circumstances attending it and the holiday season all add to the great tragedy of his passing. He was one of the best known and highly respected citizens of Athol, where his death will be keenly felt, and his widow and family have the most heartfelt sympathy of all who knew him. FRANCIS M. MCMURRAY, M.D.,

Secretary, Worcester North District Medical Society.

JONES—FREDERICK ELMER JONES, M.D., of 48 Corey Road, Brookline, Massachusetts, died suddenly December 21, 1934. He was born in 1870 at Corinna, Maine, the son of Albion K. and Elizabeth Lane Jones, graduated in medicine from the Baltimore University School of Medicine in 1897, and settled in Roxbury, later moving to Brookline.

He was a Fellow of the Massachusetts Medical Society and the American Medical Association, and a member of the Brookline Medical Club.

Dr. Jones is survived by a sister, Mrs. Merton D. Raynes, of Everett. His wife, Mrs. Florence Knox Jones, died several years ago.

WYMAN — JOHN HOWARD WYMAN, M.D., of Medway, died at the Milford Hospital, December 17, 1934.

He was born in 1879, was educated at Bowdoin College, and received his degree in medicine from the Harvard Medical School in 1906.

He was medical examiner for Millis, Bellingham, Medfield, Norfolk, and Medway, and had served his town as selectman and school physician. He was a member of the staff of the Milford Hospital, and served in the World War as Major at Camp Devens, New York, and in the South.

He is survived by his widow, Mrs. Daisy Wyman, and two sons, Hallway Wyman and Walter Wyman.

The Massachusetts Medical Society

SECOND ANNUAL POSTGRADUATE MEDICAL EXTENSION COURSE

The following session has been arranged by the Committee:

Norfolk South

Monday, January 7, at 8:30 P.M., at the Quincy City Hospital, Quincy. Subject: Dermatology and Syphilis. David L. Belding, M.D., Chairman.

NOTICES

CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P.M. on Thursday, January 3, in the Amphitheatre of the Peter Bent Brigham Hospital, Dr. Henry A. Christian, Physician-in-Chief, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, will give a medical clinic. To it are cordially invited practitioners and medical students. These clinics will be repeated on Thursdays until May.

On Saturdays in the wards of the Peter Bent Brigham Hospital, from 10 to 12, staff rounds will be conducted by Dr. Christian. These are open to all physicians.

REMOVAL

WILLIAM D. WHEELER, M.D., announces the removal of his offices to 452 Beacon Street, Boston, Massachusetts.

REPORTS AND NOTICES OF MEETINGS

THE HARVARD MEDICAL SOCIETY

A meeting of the Harvard Medical Society was held in the Peter Bent Brigham Hospital on November 27, 1934. Dr. Homans presided. The first case presented was that of a sixty-five year old Russian Jew who entered complaining of a pain in his right foot. The pain usually came on at night, and was relieved by walking or hanging his leg over the side of the bed. He was accustomed to smoking over one package of cigarettes a day. Upon examination the big toe on the right foot was blue and cold. The white count was 13,400. There was a slight trace of albumin in the urine. The oscillogram showed greater pulsations in the left foot than in the right. The man denied any attacks of intermittent claudication. Six months previous to entry there had been a prostatectomy under spinal anesthesia. Dr. Cutler suggested the possibility of a brain tumor, or the spinal anesthetic causing the condition. Dr. Allen reported having seen several cases of this sort caused by spinal anesthesia, but he believed arteriosclerosis to be the probable mechanism here.

The next patient was a fifty-four year old woman complaining of weakness and palpitation, who had had a hysterectomy six months previously under ether anesthesia. Postoperatively she had had two attacks of paroxysmal tachycardia, and following them she had a sudden pain in the left leg which turned blue. No pulsation was felt below the femoral triangle. After a vacuum pump had been used for twelve hours, the leg resumed its normal color. Thus a collateral circulation had promptly been established following a serious arterial embolism.

The third patient was a forty-nine year old man who eight years ago had had pain and discoloration of the little toe on the left foot. He also had had intermittent claudication, and two years ago developed spontaneous pains in the left leg. Physical examination showed a discolored leg and foot, the toes of which were all red and cyanosed. The dorsalis pedis was not felt on either side. Protein therapy and Buerger's exercises were instituted, but the pain recurred this last winter and he was treated with five per cent saline injections in New York. This was a case of Buerger's Disease, and it was noted that several such patients have reported marked improvement after these saline injections. Dr. Karl Walter discussed the saline treatment and said that they had given three hundred cubic centimeters of a five per cent solution to the patient, but he had not experienced any fever or reaction such as he had with the New York treatment. This brought up the question of whether the so-called saline treatments really contain some foreign protein. Dr. J. C. White said that saline does not increase the flow of blood.

The first speaker of the evening was Dr. Alfred Friedlander, Dean of the College of Medicine of the University of Cincinnati. He spoke on "Oscillometry in Medicine." The French have been very active for the past fifty years in this science. Dr. Friedlander has studied about seven hundred cases and has been especially interested in the form of the oscillogram. He first described the instrument used and said that the movements of the oscillating needle are noted at every ten millimeter drop in pressure. The patient is recumbent during the test. He presented four typical oscillograms where the oscillations were plotted against the pressure. In the normal type, oscillations began between one hundred and twenty and one hundred and forty millimeters of mercury pressure, and the highest point occurs at about one hundred millimeters, after which they fade rapidly. In the type where there is marked vascular sclerosis the curve is almost flat. In patients with malignant hypertension there is a "shift to the left", and there are oscillations even at three hundred millimeters of pressure, the maximum point being well above one hundred millimeters. In the medial or Mönckeberg's type of sclerosis, there is a great difference between the leg and thigh and between the forearm and upper arm oscillograms. Besides these typical forms, there are also mixed types. Dr. Friedlander has been able to diagnose vasospasm by inducing vasodilation by heat, and then repeating the oscillometric determination. He then showed a number of graphs in different diseases, and demonstrated how malignant hypertension with nephrosclerosis and other conditions can be differentiated.

In rheumatic heart disease, if the graph is abnormal, there is a "shift to the right" and the maximum point is below one hundred millimeters. This is not dependent on the height of the blood pressure. In luetic heart disease there is often a medial sclerosis type of curve, and the graph is very different from the rheumatic heart disease type. Therefore, the etiology of some cases of aortic regurgitation can be differentiated by this method. Luetic heart disease and hypertensive heart disease give a much more similar type of curve. Forty-two per cent of thyroid adenomas show vascular sclerosis.

The activity of tuberculosis has no effect on the oscillogram, nor is there any typical finding in coronary disease. One of the most important differentiations is between vasospasm and obliterative disease of the arteries. Bichloride of mercury causes a spasm of the arteries which is completely overcome by the administration of amyl nitrite. Tobacco in people who are not used to smoking causes a definite spasm of the arteries when three cigarettes are smoked in rapid succession. Out of thirty-three cases of diabetes, four cases gave typical curves of medial sclerosis and sixty-three per cent showed arteriosclerosis. Dr. Friedlander concluded by saying: first, that the form of the oscillogram is important; secondly, that the medial type of sclerosis can be demonstrated; thirdly, that rheumatic heart disease and luetic disease of the aorta can often be distinguished; fourthly, that there is a high instance of arteriosclerosis in diabetes; and fifthly, that there are many other possible clinical applications.

The other speaker of the evening was Dr. Louis

G. Herrmann, Assistant Professor of Surgery, University of Cincinnati, College of Medicine. He spoke on "Recent Advances in the Treatment of Obliterative Arterial Diseases of the Extremities." The objective in the treatment of these diseases is to prevent serious sequelae, chiefly gangrene. Dr. Herrmann gave a classification of arterial diseases, distinguishing a functional vasomotor type from the organic type, the latter of which he subdivided into traumatic, inflammatory, and degenerative.

In the treatment of obliterative arterial diseases the first problem is to prevent infection; the second is to establish a collateral circulation; and the third is to relieve pain. The patient with such disease must take special precautions to prevent infection. The optimum level of the foot at rest is several inches below the level of the heart, and the feet should be kept at this level at all times when they are not in use. Vascular exercises are also helpful. Certain chemical methods to increase blood flow, such as foreign protein, have been used, but pure salt has no effect.

Environmental pressure has a definite effect on the circulation. Murray in 1812 was the first to suggest a negative and positive pressure, but he did not use these at the same time. Clanny in 1832 used negative pressure, and Bluck in 1887 used alternating positive and negative pressure, because prolonged negative pressure caused stasis. This method was not revived until 1932 and 1933 when Reid and Herrmann demonstrated a striking benefit in patients with obliterative arterial disease. Seventy millimeters of positive and negative pressure were used, but there were a number of secondary thromboses, due to collapse of the venules and arterioles. Landis in 1933 used a higher positive and negative pressure with a sudden increase and decrease, in contrast to Herrmann's type of pressure curve where the increase and decrease are gradual. In 1933 Herrmann modified his method so that forty millimeters of positive and negative pressure were used and the cycle was increased to four times a minute, rather than two.

By this method Dr. Herrmann says that the skin temperature is raised eight degrees Centigrade in five minutes, that the maximum effect is procured in twenty-five minutes, and that the benefit lasts for forty-eight hours. He presented four types of cases. First, those of acute embolism, which are easy to treat, and in which he used eighty millimeters of negative pressure and forty of positive pressure. At the end of fifteen minutes, in one instance, all the pain had disappeared and, after a total of seventy hours of treatment, a sufficient collateral circulation for the entire leg below the hip had been established. Secondly, in cases of frozen feet, twenty minutes of treatment markedly relieved the symptoms, and treatment extending over four days prevented the loss of more than the tips of two toes where the entire foot had been involved. Thirdly, in cases of acute thrombosis in arteriosclerotics he completely relieved the ischemia after some hundred hours of treatment. Fourthly, in the treatment of gangrene he found that his form of therapy was most effective when the main artery was involved. When there is a true thromboangiitis obliterans, it does not help permanently, since here the trouble is in the arterioles.

Dr. Herrmann concluded his lecture by saying that much work is still needed to establish the best type of cycle, that no thrombosis will result if a low positive pressure is used, and that there is much to offer in obliterative diseases of the main arteries. He suggests as a name for this treatment "Pavex", this term being a condensation of "passive vascular exercise".

Dr. Allen discussed the paper and said that some work had been done at the Massachusetts General Hospital with Dr. Herrmann's treatment. He agrees that it offers more in peripheral embolism than surgery does, but he warned against its use where there are open lesions, as in many cases of diabetes, because of the danger of massaging the infection up the leg. Dr. Joslin pointed out the significance of the economic aspect of this form of treatment in diabetics where a great deal of time and money can be saved. He said that sixty-two per cent of diabetics die of arteriosclerosis.

The meeting was closed with a few words by Drs. Homans, Friedlander, and Herrmann.

FAULKNER HOSPITAL CLINICAL MEETING

On Thursday afternoon, December 6, the usual clinical meeting was held at the Faulkner Hospital.

Two of the cases which had come to autopsy during the past month were discussed.

One was a case of multiple myeloma, which was of unusual interest in that it was impossible to decide from an x-ray examination of the spine whether the compression of one of the vertebrae was due to new growth or to atrophy from an old fracture. The reason the latter was suspected as a probability was because four years before, the patient had had an accident in which some bones of the upper arm and shoulder region were fractured. Also in the x-ray pictures of the chest there was evidence of old fractures of many of the ribs with callus formation. Here again it was difficult to decide whether these fractures were associated with the tumor growth or associated with the trauma. The usual instructive sign which is so frequent in patients with malignant disease of the spine, namely, difficulty in sitting up in bed was present in this case. At autopsy extensive tumor formation existed throughout the bones of the sternum, ribs and vertebrae. The diseased vertebra which showed compression in the x-ray was replaced by the tumor mass, which had extended out laterally as though distorted by pressure. It turned out that despite the pain in the back and general weakness from which this patient had suffered, she had been sitting up with the hope that it would improve her condition, although this procedure was extremely painful. It was felt

that possibly a mechanical factor had contributed to the distortion of the vertebra as shown by x-ray.

The other case was a typical case of chronic renal disease called by the pathologist benign nephrosclerosis. In addition there was hypertension with a cerebral hemorrhage as the immediate cause of death. The patient also had mild diabetes and there were vascular lesions in the pancreas similar to the vascular lesions in the kidney. The vascular lesion prominent in the afferent arterioles leading to the glomeruli was very prettily demonstrated in the microscopic section. It seems exceedingly unfortunate that this term nephrosclerosis which the pathologists are now using quite generally does not even appear in the index of one of the most popular textbooks of medicine. It is about time that a conscientious effort was made on the part of the clinicians to speak of nephritis in the same terms that the pathologists use. It seems as though recently there was a tendency to swing away from trying to classify nephritis upon a pathological basis and to use simply clinical terms as chronic nephritis with or without edema and acute hemorrhagic nephritis. The pathologists at the present time give a very clear-cut picture of what happens in the kidney and it is hoped that the clinicians will make a more serious effort to adjust their nomenclature to the pathological picture as is done in most of the other diseases.

Following the presentation of these two cases Dr. John B. Hazard, our pathologist, reported on Erythema Arthriticum Epidemicum, popularly called Haverhill fever. His report was based upon an isolated case of the disease which had developed in a medical student and had been studied in his former laboratory. He called attention to the fact that the organism which produces this disease has been isolated in France and also in this country and both seem to be identical. The organism can be obtained from the blood and from the joints and is not difficult to cultivate. It is one of those organisms which changes its form from time to time and can produce in animals conjunctivitis, pericarditis and peritonitis. He called attention to the symptoms of the disease which have been so well described by Dr. Place. These are chills and fever, joint pains and a rash. The prognosis in all cases seems to be good. The disease apparently can be transmitted by milk, although the bacteria will not grow in a media made up of milk. It seems as though it might be transmitted by the bite of a rat. Whether antibodies develop as a result of infection which will persist for any appreciable time Dr. Hazard hopes to determine by further studies on the medical students.

WILLIAM HARVEY SOCIETY

A meeting of the William Harvey Society was held on December 14, 1934, in the auditorium of the Beth Israel Hospital. Dr. Harold E. MacMahon called the meeting to order, and introduced Dr. Fuller Albright, who spoke on "Parathyroid Disease." Doctor Albright began his interesting lecture by considering the body as a bag of water with bones forming the structural part of this bag. Calcium and phosphate ions are always present in the fluid around the bones, and are constantly going in and out of these structures. The most important outlet of calcium from the body is through the kidneys, and the intake is entirely through the gastro-intestinal tract.

Osteoclasts carry away bone débris after the calcium and phosphate have been removed, and osteoblasts lay down new osteoid tissue. An enzyme called phosphatase is secreted by the osteoblasts, and has the function of changing organic phosphorus compounds into inorganic phosphate to be used in the calcification of bone. The normal blood calcium is about ten milligrams per cent, phosphorus four milligrams per cent, and phosphatase four Bodansky units per cubic centimeter of plasma.

In classical hyperparathyroidism with von Recklinghausen's Disease there is a high blood calcium, a low blood phosphate, a high urine calcium, a high urine phosphate, and a high blood phosphatase. The bones tend to be depleted of their calcium and phosphorus, and both the osteoclasts and osteoblasts are numerous, the total turnover of calcium and phosphorus through the bone being rapid, and the net result being osteoporosis, unless the calcium intake is sufficient to overcome the rapid loss through the urine. There is also a fibrosis of the bone marrow. In this condition osteoclastic masses called brown tumors, as well as cysts, may develop in the bones.

Doctor Albright then showed a series of slides which demonstrated various forms of the disease as well as certain conditions which may simulate hyperparathyroidism. A mild osteoporotic form without cystic or brown tumor formation may only lead to pain in the back. Multiple kidney stones with or without bone disease are a common manifestation. In some cases with a high calcium intake there is a shunt between the gastro-intestinal tract and the kidneys without the calcium going through the bones. In another type, calcium and phosphorus are deposited in the kidney parenchyma, leading to small sclerosed kidneys with a typical picture of Bright's Disease, while the amounts of calcium and phosphorus in the blood and urine may be normal. Calcium casts are often formed in the kidney tubules, and may be seen in the sediment.

Senile osteoporosis has a normal blood calcium, phosphorus, and phosphatase, and is due to lack of osteoblastic activity. A somewhat similar condition holds in osteogenesis imperfecta. In osteomalacia the body fluids contain so little calcium and phosphorus that they are not deposited normally, and the bone shows wide osteoid seams. This condition is often present in steatorrhea. The blood phosphatase is high.

If the tumor is removed in the presence of much bone disease, tetany is apt to develop postoperatively. After operation the osteoclasts disappear, but the osteoblasts remain increased, and the phospha-

tase may even rise. This leads to an actual osteomalacia, a low blood calcium, and tetany. When the phosphatase is high in hyperparathyroidism, tetany will follow operation. Doctor Albright took up Paget's Disease briefly, and then reviewed the twenty-five cases of hyperparathyroidism that have been treated at the Massachusetts General Hospital.

Dr. Walter Bauer discussed one patient with marked generalized osteoporosis without tumor or cyst formation on whom careful metabolic and histologic studies were made before and after operation. Previous to the removal of the parathyroid tumor, the phosphatase was thirty-two units. Within a few hours after operation the urine calcium and phosphorus fell markedly and on the fourth day the serum calcium had fallen to eight milligrams per cent. At this time the first sign of tetany developed, and the patient was shifted to a high calcium diet containing one gram of calcium per day. The phosphatase remained up, and a biopsy taken seven days postoperatively showed a decrease in the fibrous tissue, and wide osteoid bands with almost a complete cessation of osteoclastic activity. From a metabolic point of view, the bone after operation showed a very marked affinity for calcium and phosphorus, and was still packing it away nine months later.

Dr. Edward D. Churchill discussed the surgical treatment of cases of hyperparathyroidism, and pointed out the frequent difficulty of finding not only the tumors, but also the normal glands. He showed a series of slides which demonstrated both the normal and abnormal positions. Usually one is found at the end of the tiny vascular branches of the inferior thyroid artery lying on the anterior surface of the lower poles of the thyroid. The recurrent laryngeal nerve often runs close to these structures, and it must not be damaged. The tumor usually consists of an adenoma of one of the parathyroids, but occasionally they are multiple, and he has seen five cases of a diffuse generalized hyperplasia, a condition in which a subtotal parathyroidectomy is necessary. Out of twenty-one cases two adenomas were found behind the esophagus, one behind the trachea, two in the anterior mediastinum, and one in the superior mediastinum.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Van Dyke Street entrance) Tuesday evening, January 8 at 8:15 P.M.

PROGRAM

"Forty Years of Diabetes" with Presentation of Cases. By Dr. Elliott P. Joslin.

MARSHALL N. FULTON, M.D., *Secretary.*

BOSTON CITY HOSPITAL HOUSE OFFICERS' ASSOCIATION

The next meeting of the House Officers' Association of the Boston City Hospital will be held on Thursday, January 3, in the Thorndike Memorial Amphitheatre at 7:30 P.M.

PROGRAM

"The Anatomical Diagnosis of Various Types of Chronic Arthritis." By Dr. Chester Keefer.

"The Orthopedic Treatment of Chronic Arthritis." By Dr. G. E. Haggart.

ROGER S. MITCHELL, *Secretary,*
House Officers' Association.

FAULKNER HOSPITAL CLINICAL MEETING

The next meeting will be held at the Faulkner Hospital on Thursday afternoon, January 3, at 5:00 P.M. In addition to the usual clinical pathological conference on the cases which have come to autopsy during the month Dr. John S. Hodgson will give a short talk on some neurological subject. All physicians are invited.

SOCIETY MEETINGS
CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, DECEMBER 31, 1934

Thursday, January 3—

*12 M. Clinico-Pathological Conference. Massachusetts General Hospital. Pathological Building.
†12 M. Clinico-Pathological Conference. Children's Hospital Amphitheatre.
*3:30 P. M. Medical Clinic. Dr. Christian. Peter Bent Brigham Hospital Amphitheatre.
†4:30 P. M. Surgical Clinic. Children's Hospital Amphitheatre.
*5 P.M. Faulkner Hospital Clinical Meeting.
7:30 P.M. House Officers' Association, Boston City Hospital.

Friday, January 4—

†12 M. Clinical Meeting of Children's Medical Staff, Massachusetts General Hospital Ether Dome.

Saturday, January 5—

*10-12. Medical Staff Rounds. Dr. Christian. Peter Bent Brigham Hospital.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

January 3—Clinic at the Peter Bent Brigham Hospital. See page 1227.

January 3—Faulkner Hospital Clinical Meeting. See notice above.

January 3—Boston City Hospital House Officers' Association. See notice elsewhere on this page.

January 7—New England Heart Association will meet at the Peter Bent Brigham Hospital at 8:15 P.M. For details address the Secretary, Dr. James M. Faulkner, 264 Beacon Street, Boston, Mass.

January 8—Harvard Medical Society. See notice elsewhere on this page.

MASSACHUSETTS DIETETIC ASSOCIATION

January 8—Tuesday, 8 P.M. "Careers and Natural Abilities," Professor Johnson O'Connor, Human Engineering Laboratory, Stevens Institute of Technology, Hoboken, N. J.

February 12—Tuesday, 8 P.M. "Diabetic Children," Dr. Priscilla White, Joslin Diabetic Unit.

March 12—Tuesday, 8 P.M. "The Effect of Diet on Anemia," Dr. Lewis Diamond, Instructor in Medicine, Harvard University Medical School, Associate Physician, Children's Hospital.

March 19—Tuesday, 2 P.M. Field Trip: Visit Storehouse, First National Stores.

April 9—Tuesday, 8 P.M. "Small Hospital Problems," Miss Margaret Copeland, Superintendent, Free Hospital for Women.

January 11—William Harvey Society. Speaker: Dr. Charles F. McKhann. Subject: "Immune Substances in Placental Extract."

April 29 - May 3, 1935—The American College of Physicians will meet at Philadelphia. For information address Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

June, 1935—Medical Library Association will meet in Rochester, N. Y. For details address the Secretary: Miss Frances N. A. Whitman, Librarian, Harvard University Schools of Medicine and Public Health, Boston, Mass.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

The Semi-Annual Meeting will be held in January and the Annual Meeting in May, time, place and subject to be announced.

E. S. BAGNALL, M.D., Secretary.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings will be held on the second Tuesday of January, March, and May at the Weldon Hotel, Greenfield, Mass.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

January 9, 1935—Melrose.
March 13, 1935—Wakefield.
May 8, 1935—Winchester.

K. L. MACLACHLAN, M.D., Secretary.
1 Bellevue Street, Melrose.

PLYMOUTH DISTRICT MEDICAL SOCIETY

January—Goddard Hospital.
March—Plymouth County Hospital.
April—Lakeville Sanatorium.

SUFFOLK DISTRICT MEDICAL SOCIETY

January 23, 1935—General Meeting in association with the Boston Medical Library. Speaker and subject to be announced later.

March 27, 1935—Clinical Meeting at the Boston Lying-In Hospital.

April 24, 1935—Clinical Meeting at the Children's Hospital.

The medical profession is cordially invited to attend all of these meetings.

ROBERT L. DeNORMANDIE, M.D., President.
GEORGE P. REYNOLDS, M.D., Secretary.
HENRY T. HUTCHINS, M.D., Boston Medical Library.

WORCESTER DISTRICT MEDICAL SOCIETY

January 9, 1935—Wednesday evening. Worcester City Hospital, Worcester, Mass. 6:30 P.M.: Buffet supper. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Buffet supper complimentary by the Hospital.

February 13, 1935—Wednesday evening. Worcester State Hospital, Worcester, Mass. 6:30 P.M.: Dinner. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Dinner complimentary by the Hospital.

March 13, 1935—Wednesday evening. The Memorial Hospital, Worcester, Mass. 6:30 P.M.: Buffet supper. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Buffet supper complimentary by the Hospital.

April 10, 1935—Wednesday evening. Worcester Hahnemann Hospital, Worcester, Mass. 6:30 P.M.: Dinner. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Dinner complimentary by the Hospital.

May 8, 1935—Wednesday afternoon and evening. Annual Meeting of the Worcester District Medical Society. The time and place of this meeting will be announced later.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

BOOK REVIEW

Facts and Theories of Psychoanalysis. By Ives Hendrick, M.D. First Edition. 308 pp. New York. Alfred A. Knopf. $3.00.

With all the books that have appeared on the general subject of psychoanalysis one might easily wonder whether another could be produced with new values for any class of readers. And yet it seems that Dr. Hendrick has achieved this result by writing a particularly informative epitome covering the whole field of psychoanalysis. The author states that this was the aim which he set himself, and he has not fallen far short of his mark.

The reviewer tends to disagree with the cover advertisement which implies that the book is particularly addressed to the layman and to those who have engaged in no previous study of psychoanalysis. On the contrary, it is much more likely to be serviceable to serious students who have already been introduced to this subject, preferably by such works as Freud's "Introductory Lectures." Written by a practicing psychiatrist and analyst, the book is especially suitable for psychiatrists and physicians who wish to orient themselves concerning the relationships of psychoanalysis to the problems that neurologists and psychiatrists have to meet. On the other hand, the author specifically states that it is not a handbook of instruction in technique; indeed, in his section on psychoanalytic education he lays great stress on the special training that is to be acquired only through "didactic analyses".

The book is intelligently divided into three main sections: I. The Facts of Psychoanalysis, II. The Theories of Psychoanalysis, III. Therapy by Psychoanalysis. The last includes discussion of psychoanalytic method and indications for psychoanalytic therapy, matters of profound interest to physicians.

Throughout, the author shows his sound knowledge of theory and his good grounding in technical application of theory. Moreover he utilizes his own clinical experience. The topical method of presentation is followed and is so organized that the reader is able to select for his own needs succinct statements presenting the various concepts of psychoanalytic theory and practice. Vignettes of case material are frequently interspersed to illuminate the subject in hand.

In spite of the topical headings which incline the reader to select for perusal chapters and sections of special interest, the book is one to be read consecutively because fragments of the same case appear from time to time. Significant points may be missed if earlier statements about the same case have not been read.

The reviewer could easily indulge in some adverse criticism, bringing up questions of grammar, unevenness and carelessness in writing, and a few unclear statements. And he wishes once again that the ghostly myth about witches being burned—burning somehow does seem worse than hanging—could be laid once and for all. But these are very minor matters and quite beside the point that the book in general is the embodiment of clear-cut statements, and that it is thoroughly readable and immensely informative for those who care to look into a branch of psychological medicine which is making an immense impression upon students of mental life, whether they are interested in the abnormal or normal.

The pages that deal with "Personality Potential" as related to treatment possibilities evidently represent the author's own thinking upon this important matter. These sections, together with what little is said about the concern of psychoanalysis with certain organic ailments such as gastric ulcer, are particularly to be recommended for reading by physicians.

The last twenty pages of the text are devoted to a glossary, the compilation of which could not have been an easy matter. One might cavil about a few of the definitions; and why some common, well-known words were defined and other less-known, important terms were not given more space seems inexplicable. But while it could be bettered in a later edition, as a whole the glossary is sound and serviceable.